

Appendix A

Substances for which Emissions Must Be Quantified

(as of August, 2007)

SUBSTANCES FOR WHICH EMISSIONS MUST BE QUANTIFIED

CAS number	Substance name
75070	Acetaldehyde
60355	Acetamide
75058	Acetonitrile
98862	Acetophenone
53963	2-Acetylaminofluorene [PAH-Derivative, POM]
107028	Acrolein
79061	Acrylamide
79107	Acrylic acid
107131	Acrylonitrile
107051	Allyl chloride
7429905	Aluminum
1344281	Aluminum oxide (fibrous forms)
117793	2-Aminoanthraquinone [PAH-Derivative, POM]
92671	4-Aminobiphenyl [POM]
61825	Amitrole
7664417	Ammonia
6484522	Ammonium nitrate
7783202	Ammonium sulfate
62533	Aniline
90040	o-Anisidine
-	Anthracene [PAH, POM], (see PAH)
7440360	Antimony
*	Antimony compounds including but not limited to:
1309644	Antimony trioxide
7440382	Arsenic
1016	Arsenic compounds (inorganic) including but not limited to:
7784421	Arsine
1017	Arsenic compounds (other than inorganic)
-	Asbestos (see Mineral fibers)
7440393	Barium
*	Barium Compounds
-	Benz[a]anthracene [PAH, POM], (see PAH)
71432	Benzene
92875	Benzidine (and its salts) [POM]
1020	Benzidine-based dyes [POM] including but not limited to:
1937377	Direct Black 38 [PAH-Derivative, POM]
2602462	Direct Blue 6 [PAH-Derivative, POM]
16071866	Direct Brown 95 (technical grade) [POM]
-	Benzo[a]pyrene [PAH, POM], (see PAH)
-	Benzo[b]fluoranthene [PAH, POM], (see PAH)
271896	Benzofuran
98077	Benzoic trichloride {Benzotrichloride}
-	Benzo[j]fluoranthene [PAH, POM] (see PAH)
-	Benzo[k]fluoranthene [PAH, POM] (see PAH)
98884	Benzoyl chloride
94360	Benzoyl peroxide

CAS number	Substance name
100447	Benzyl chloride
7440417	Beryllium
*	Beryllium compounds
92524	Biphenyl [POM]
111444	Bis(2-chloroethyl) ether {DCEE}
542881	Bis(chloromethyl) ether
103231	Bis(2-ethylhexyl) adipate
7726956	Bromine
*	Bromine compounds (inorganic) including but not limited to:
7789302	Bromine pentafluoride
10035106	Hydrogen bromide
7758012	Potassium bromate
75252	Bromoform
106990	1,3-Butadiene
540885	t-Butyl acetate
141322	Butyl acrylate
71363	n-Butyl alcohol
78922	sec-Butyl alcohol
75650	tert-Butyl alcohol
85687	Butyl benzyl phthalate
7440439	Cadmium
*	Cadmium compounds
156627	Calcium cyanamide
105602	Caprolactam
2425061	Captafol
133062	Captan
63252	Carbaryl [PAH-Derivative, POM]
1050	Carbon black extracts
75150	Carbon disulfide
56235	Carbon tetrachloride
463581	Carbonyl sulfide
1055	Carrageenan (degraded)
120809	Catechol
133904	Chloramben
57749	Chlordane
108171262	Chlorinated paraffins (average chain length, C12; approximately 60% Chlorine by weight)
7782505	Chlorine
10049044	Chlorine dioxide
79118	Chloroacetic acid
532274	2-Chloroacetophenone
106478	p-Chloroaniline
1058	Chlorobenzenes including but not limited to:
108907	Chlorobenzene
25321226	Dichlorobenzenes (mixed isomers) including:
95501	1,2-Dichlorobenzene
541731	1,3-Dichlorobenzene
106467	p-Dichlorobenzene {1,4-Dichlorobenzene}
120821	1,2,4-Trichlorobenzene
510156	Chlorobenzilate [POM] {Ethyl-4,4'-dichlorobenzilate}

CAS number	Substance name
67663	Chloroform
107302	Chloromethyl methyl ether (technical grade)
1060	Chlorophenols including but not limited to:
95578	2-Chlorophenol
120832	2,4-Dichlorophenol
87865	Pentachlorophenol
25167833	Tetrachlorophenols including but not limited to:
58902	2,3,4,6-Tetrachlorophenol
95954	2,4,5-Trichlorophenol
88062	2,4,6-Trichlorophenol
95830	4-Chloro-o-phenylenediamine
76062	Chloropicrin
126998	Chloroprene
95692	p-Chloro-o-toluidine
7440473	Chromium
*	Chromium compounds (other than hexavalent)
18540299	Chromium, hexavalent (and compounds) including but not limited to:
10294403	Barium chromate
13765190	Calcium chromate
1333820	Chromium trioxide
7758976	Lead chromate
10588019	Sodium dichromate
7789062	Strontium chromate
-	Chrysene [PAH, POM], (see PAH)
7440484	Cobalt
*	Cobalt compounds
1066	Coke oven emissions
7440508	Copper
*	Copper compounds
1070	Creosotes
120718	p-Cresidine
1319773	Cresols (mixtures of) {Cresylic acid} including:
108394	m-Cresol
95487	o-Cresol
106445	p-Cresol
4170303	Crotonaldehyde
98828	Cumene
80159	Cumene hydroperoxide
135206	Cupferron
1073	Cyanide compounds (inorganic) including but not limited to:
74908	Hydrocyanic acid
110827	Cyclohexane
108930	Cyclohexanol
66819	Cycloheximide
	Decabromodiphenyl oxide [POM] (see Polybrominated diphenyl ethers)
1075	Dialkylnitrosamines including but not limited to:
924163	N-Nitrosodi-n-butylamine
1116547	N-Nitrosodiethanolamine
55185	N-Nitrosodiethylamine
62759	N-Nitrosodimethylamine

CAS number	Substance name
621647	N-Nitrosodi-n-propylamine
10595956	N-Nitrosomethylethylamine
615054	2,4-Diaminoanisole
1078	Diaminotoluenes (mixed isomers) including but not limited to:
95807	2,4-Diaminotoluene {2,4-Toluene diamine}
334883	Diazomethane
226368	Dibenz[a,h]acridine [POM]
224420	Dibenz[a,j]acridine [POM]
-	Dibenz[a,h]anthracene [PAH, POM], (see PAH)
194592	7H-Dibenzo[c,g]carbazole
-	Dibenzo[a,e]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,h]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,i]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,l]pyrene [PAH, POM], (see PAH)
132649	Dibenzofuran [POM]
96128	1,2-Dibromo-3-chloropropane {DBCP}
96139	2,3-Dibromo-1-propanol
84742	Dibutyl phthalate
-	p-Dichlorobenzene (1,4-Dichlorobenzene) (see Chlorobenzenes)
91941	3,3'-Dichlorobenzidine [POM]
72559	Dichlorodiphenyldichloroethylene {DDE} [POM]
75343	1,1-Dichloroethane {Ethylidene dichloride}
94757	Dichlorophenoxyacetic acid, salts and esters {2,4-D}
78875	1,2-Dichloropropane {Propylene dichloride}
542756	1,3-Dichloropropene
62737	Dichlorovos {DDVP}
115322	Dicofol [POM]
--	Diesel engine exhaust
9901	Diesel engine exhaust, particulate matter {Diesel PM}
9902	Diesel engine exhaust, total organic gas
#	Diesel fuel (marine)
111422	Diethanolamine
117817	Di(2-ethylhexyl) phthalate {DEHP}
64675	Diethyl sulfate
119904	3,3'-Dimethoxybenzidine [POM]
60117	4-Dimethylaminoazobenzene [POM]
121697	N,N-Dimethylaniline
57976	7,12-Dimethylbenz[a]anthracene [PAH-Derivative, POM]
119937	3,3'-Dimethylbenzidine {o-Tolidine} [POM]
79447	Dimethyl carbamoyl chloride
68122	Dimethyl formamide
57147	1,1-Dimethylhydrazine
131113	Dimethyl phthalate
77781	Dimethyl sulfate
534521	4,6-Dinitro-o-cresol (and salts)
51285	2,4-Dinitrophenol
42397648	1,6-Dinitropyrene [PAH-Derivative, POM]
42397659	1,8-Dinitropyrene [PAH-Derivative, POM]
25321146	Dinitrotoluenes (mixed isomers) including but not limited to:
121142	2,4-Dinitrotoluene

CAS number	Substance name
606202	2,6-Dinitrotoluene
123911	1,4-Dioxane
-	Dioxins (Chlorinated dibenzodioxins) (see Polychlorinated dibenzo-p-dioxins) [POM]
630933	Diphenylhydantoin [POM]
122667	1,2-Diphenylhydrazine {Hydrazobenzene} [POM]
1090	Environmental Tobacco Smoke
106898	Epichlorohydrin
106887	1,2-Epoxybutane
1091	Epoxy resins
140885	Ethyl acrylate
100414	Ethyl benzene
75003	Ethyl chloride {Chloroethane}
-	Ethyl-4,4'-dichlorobenzilate (see Chlorobenzilate)
74851	Ethylene
106934	Ethylene dibromide {EDB, 1,2-Dibromoethane}
107062	Ethylene dichloride {EDC, 1,2-Dichloroethane}
107211	Ethylene glycol
151564	Ethyleneimine {Aziridine}
75218	Ethylene oxide
96457	Ethylene thiourea
1101	Fluorides and compounds including but not limited to:
7664393	Hydrogen fluoride
1103	Fluorocarbons (brominated)
1104	Fluorocarbons (chlorinated) including but not limited to:
76131	Chlorinated fluorocarbon {CFC-113} {1,1,2-Trichloro-1,2,2-trifluoroethane}
75456	Chlorodifluoromethane {Freon 22}
75718	Dichlorodifluoromethane {Freon 12}
75434	Dichlorofluoromethane {Freon 21}
75694	Trichlorofluoromethane {Freon 11}
50000	Formaldehyde
110009	Furan
--	Gasoline engine exhaust including but not limited to:
--	Gasoline engine exhaust (condensates & extracts)
9910	Gasoline engine exhaust, particulate matter
9911	Gasoline engine exhaust, total organic gas
1110	Gasoline vapors
111308	Glutaraldehyde
1115	Glycol ethers and their acetates including but not limited to:
111466	Diethylene glycol
111966	Diethylene glycol dimethyl ether
112345	Diethylene glycol monobutyl ether
111900	Diethylene glycol monoethyl ether
111773	Diethylene glycol monomethyl ether
25265718	Dipropylene glycol
34590948	Dipropylene glycol monomethyl ether
629141	Ethylene glycol diethyl ether
110714	Ethylene glycol dimethyl ether
111762	Ethylene glycol monobutyl ether

CAS number	Substance name
110805	Ethylene glycol monoethyl ether
111159	Ethylene glycol monoethyl ether acetate
109864	Ethylene glycol monomethyl ether
110496	Ethylene glycol monomethyl ether acetate
2807309	Ethylene glycol monopropyl ether
107982	Propylene glycol monomethyl ether
108656	Propylene glycol monomethyl ether acetate
112492	Triethylene glycol dimethyl ether
76448	Heptachlor
118741	Hexachlorobenzene
87683	Hexachlorobutadiene
608731	Hexachlorocyclohexanes (mixed or technical grade) including but not limited to:
319846	alpha-Hexachlorocyclohexane
319857	beta-Hexachlorocyclohexane
58899	Lindane {gamma-Hexachlorocyclohexane }
77474	Hexachlorocyclopentadiene
67721	Hexachloroethane
680319	Hexamethylphosphoramide
110543	Hexane
302012	Hydrazine
7647010	Hydrochloric acid
-	Hydrocyanic acid (see Cyanide compounds)
7783064	Hydrogen sulfide
123319	Hydroquinone
-	Indeno[1,2,3-cd]pyrene [PAH, POM], (see PAH)
13463406	Iron pentacarbonyl
1125	Isocyanates including but not limited to:
822060	Hexamethylene-1,6-diisocyanate
101688	Methylene diphenyl diisocyanate {MDI} [POM]
624839	Methyl isocyanate
-	Toluene-2,4-diisocyanate (see Toluene diisocyanates)
-	Toluene-2,6-diisocyanate (see Toluene diisocyanates)
78591	Isophorone
78795	Isoprene, except from vegetative emission sources
67630	Isopropyl alcohol
80057	4,4'-Isopropylidenediphenol [POM]
7439921	Lead
1128	Lead compounds (inorganic) including but not limited to:
301042	Lead acetate
-	Lead chromate (see Chromium, hexalent)
7446277	Lead phosphate
1335326	Lead subacetate
1129	Lead compounds (other than inorganic)
108316	Maleic anhydride
7439965	Manganese
*	Manganese compounds
7439976	Mercury
*	Mercury compounds including but not limited to:
7487947	Mercuric chloride

CAS number	Substance name
593748	Methyl mercury {Dimethylmercury}
67561	Methanol
72435	Methoxychlor [POM]
75558	2-Methylaziridine {1,2-Propyleneimine}
74839	Methyl bromide {Bromomethane}
74873	Methyl chloride {Chloromethane}
71556	Methyl chloroform {1,1,1-Trichloroethane}
56495	3-Methylcholanthrene [PAH-Derivative, POM]
3697243	5-Methylchrysene [PAH-Derivative, POM]
101144	4,4'-Methylene bis(2-chloroaniline) {MOCA} [POM]
75092	Methylene chloride {Dichloromethane}
101779	4,4'-Methylenedianiline (and its dichloride) [POM]
78933	Methyl ethyl ketone {2-Butanone}
60344	Methyl hydrazine
74884	Methyl iodide {Iodomethane}
108101	Methyl isobutyl ketone {Hexone}
75865	2-Methylactonitrile {Acetone cyanohydrin}
80626	Methyl methacrylate
109068	2-Methylpyridine
1634044	Methyl tert-butyl ether
90948	Michler's ketone [POM]
1136	Mineral fibers (fine mineral fibers which are man-made, and are airborne particles of a respirable size greater than 5 microns in length, less than or equal to 3.5 microns in diameter, with a length to diameter ratio of 3:1) including but not limited to:
1056	Ceramic fibers
1111	Glasswool fibers
1168	Rockwool
1181	Slagwool
1135	Mineral fibers (other than man-made) including but not limited to:
1332214	Asbestos
12510428	Erionite
1190	Talc containing asbestiform fibers
1313275	Molybdenum trioxide
-	Naphthalene [PAH, POM], (see PAH)
7440020	Nickel
*	Nickel compounds including but not limited to:
373024	Nickel acetate
3333673	Nickel carbonate
13463393	Nickel carbonyl
12054487	Nickel hydroxide
1271289	Nickelocene
1313991	Nickel oxide
12035722	Nickel subsulfide
1146	Nickel refinery dust from the pyrometallurgical process
7697372	Nitric acid
139139	Nitrilotriacetic acid
602879	5-Nitroacenaphthene [PAH-Derivative, POM]
98953	Nitrobenzene
92933	4-Nitrobiphenyl [POM]

CAS number	Substance name
7496028	6-Nitrochrysene [PAH-Derivative, POM]
607578	2-Nitrofluorene [PAH-Derivative, POM]
302705	Nitrogen mustard N-oxide
100027	4-Nitrophenol
79469	2-Nitropropane
5522430	1-Nitropyrene [PAH-Derivative, POM]
57835924	4-Nitropyrene [PAH-Derivative, POM]
86306	N-Nitrosodiphenylamine
156105	p-Nitrosodiphenylamine [POM]
684935	N-Nitroso-N-methylurea
59892	N-Nitrosomorpholine
100754	N-Nitrosopiperidine
930552	N-Nitrosopyrrolidine
*	Oleum (see Sulfuric acid and oleum)
--	PAHs (Polycyclic aromatic hydrocarbons) [POM] including but not limited to:
1151	PAHs, total, w/o individ. components reported [PAH, POM]
1150	PAHs, total, with individ. components also reported [PAH, POM]
83329	Acenaphthene [PAH, POM]
208968	Acenaphthylene [PAH, POM]
120127	Anthracene [PAH, POM]
56553	Benz[a]anthracene [PAH, POM]
50328	Benzo[a]pyrene [PAH, POM]
205992	Benzo[b]fluoranthene
192972	Benzo[e]pyrene [PAH, POM]
191242	Benzo[g,h,i]perylene [PAH, POM]
205823	Benzo[j]fluoranthene [PAH, POM]
207089	Benzo[k]fluoranthene [PAH, POM]
218019	Chrysene [PAH, POM]
53703	Dibenz[a,h]anthracene [PAH, POM]
192654	Dibenzo[a,e]pyrene [PAH, POM]
189640	Dibenzo[a,h]pyrene [PAH, POM]
189559	Dibenzo[a,i]pyrene [PAH, POM]
191300	Dibenzo[a,l]pyrene [PAH, POM]
206440	Fluoranthene [PAH, POM]
86737	Fluorene [PAH, POM]
193395	Indeno[1,2,3-cd]pyrene [PAH, POM]
91576	2-Methyl naphthalene [PAH, POM]
91203	Naphthalene [PAH, POM]
198550	Perylene [PAH, POM]
85018	Phenanthrene [PAH, POM]
129000	Pyrene [PAH, POM]
#	PAH-Derivatives (Polycyclic aromatic hydrocarbon derivatives) [POM] (including but not limited to those substances listed in Appendix A with the bracketed designation [PAH-Derivative, POM])
56382	Parathion
1336363	PCBs (Polychlorinated biphenyls), total [POM] including but not limited to:
32598133	3,3',4,4'-Tetrachlorobiphenyl (PCB 77)
70362504	3,4,4',5-Tetrachlorobiphenyl (PCB 81)
32598144	2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)

CAS number	Substance name
74472370	2,3,4,4',5-Pentachlorobiphenyl (PCB 114)
31508006	2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
65510443	2,3',4,4',5'-Pentachlorobiphenyl (PCB 123)
57465288	3,3',4,4',5-Pentachlorobiphenyl (PCB 126)
38380084	2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
69782907	2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)
52663726	2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
32774166	3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)
39635319	2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)
82688	Pentachloronitrobenzene {Quintobenzene}
79210	Peracetic acid
127184	Perchloroethylene {Tetrachloroethene}
2795393	Perfluorooctanoic acid {PFOA} and its salts, esters, and sulfonates
108952	Phenol
106503	p-Phenylenediamine
90437	2-Phenylphenol [POM]
75445	Phosgene
7723140	Phosphorus
--	Phosphorus compounds:
7803512	Phosphine
7664382	Phosphoric acid
10025873	Phosphorus oxychloride
10026138	Phosphorus pentachloride
1314563	Phosphorus pentoxide
7719122	Phosphorus trichloride
126738	Tributyl phosphate
78400	Triethyl phosphine
512561	Trimethyl phosphate
78308	Triorthocresyl phosphate [POM]
115866	Triphenyl phosphate [POM]
101020	Triphenyl phosphite [POM]
85449	Phthalic anhydride
2222	Polybrominated diphenyl ethers {PBDEs}, including but not limited to:
1163195	Decabromodiphenyl oxide [POM]
--	Polychlorinated dibenzo-p-dioxins {PCDDs or Dioxins} [POM] including but not limited to:
1086	Dioxins, total, w/o individ. isomers reported {PCDDs} [POM]
1085	Dioxins, total, with individ. isomers also reported {PCDDs} [POM]
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin {TCDD} [POM]
40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin [POM]
39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin [POM]
57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin [POM]
19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin [POM]
35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin [POM]
3268879	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin [POM]
41903575	Total Tetrachlorodibenzo-p-dioxin [POM]
36088229	Total Pentachlorodibenzo-p-dioxin [POM]
34465468	Total Hexachlorodibenzo-p-dioxin [POM]
37871004	Total Heptachlorodibenzo-p-dioxin [POM]

CAS number	Substance name
--	Polychlorinated dibenzofurans {PCDFs or Dibenzofurans} [POM] including but not limited to:
1080	Dibenzofurans (Polychlorinated dibenzofurans) {PCDFs} [POM]
51207319	2,3,7,8-Tetrachlorodibenzofuran [POM]
57117416	1,2,3,7,8-Pentachlorodibenzofuran [POM]
57117314	2,3,4,7,8-Pentachlorodibenzofuran [POM]
70648269	1,2,3,4,7,8-Hexachlorodibenzofuran [POM]
57117449	1,2,3,6,7,8-Hexachlorodibenzofuran [POM]
72918219	1,2,3,7,8,9-Hexachlorodibenzofuran [POM]
60851345	2,3,4,6,7,8-Hexachlorodibenzofuran [POM]
67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran [POM]
55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran [POM]
39001020	1,2,3,4,6,7,8,9-Octachlorodibenzofuran [POM]
55722275	Total Tetrachlorodibenzofuran [POM]
30402154	Total Pentachlorodibenzofuran [POM]
55684941	Total Hexachlorodibenzofuran [POM]
38998753	Total Heptachlorodibenzofuran [POM]
#	POM (Polycyclic organic matter) (including but not limited to those substances listed in Appendix A with the bracketed designation of [POM], [PAH, POM], or [PAH-Derivative, POM])
1120714	1,3-Propane sultone
57578	beta-Propiolactone
123386	Propionaldehyde
114261	Propoxur {Baygon}
115071	Propylene
75569	Propylene oxide
-	1,2-Propyleneimine (see 2-Methylaziridine)
110861	Pyridine
91225	Quinoline
106514	Quinone
1165	Radionuclides including but not limited to:
24267569	Iodine-131
1166	Radon and its decay products
50555	Reserpine [POM]
#	Residual (heavy) fuel oils
7782492	Selenium
*	Selenium compounds including but not limited to:
7783075	Hydrogen selenide
7446346	Selenium sulfide
1175	Silica, crystalline (respirable)
7440224	Silver
*	Silver compounds
1310732	Sodium hydroxide
100425	Styrene
96093	Styrene oxide
*	Sulfuric acid and oleum
8014957	Oleum
7446719	Sulfur trioxide
7664939	Sulfuric acid
100210	Terephthalic acid

CAS number	Substance name
79345	1,1,2,2-Tetrachloroethane
-	Tetrachlorophenols (see Chlorophenols)
7440280	Thallium
*	Thallium compounds
62555	Thioacetamide
62566	Thiourea
7550450	Titanium tetrachloride
108883	Toluene
-	2,4-Toluenediamine (see 2,4-Diaminotoluene)
26471625	Toluene diisocyanates including but not limited to:
584849	Toluene-2,4-diisocyanate
91087	Toluene-2,6-diisocyanate
95534	o-Toluidine
8001352	Toxaphene {Polychlorinated camphenes}
-	1,1,1-Trichloroethane (see Methyl chloroform)
79005	1,1,2-Trichloroethane {Vinyl trichloride}
79016	Trichloroethylene
-	2,4,6-Trichlorophenol (see Chlorophenols)
96184	1,2,3-Trichloropropane
121448	Triethylamine
1582098	Trifluralin
25551137	Trimethylbenzenes including but not limited to:
95636	1,2,4-Trimethylbenzene
540841	2,2,4-Trimethylpentane
51796	Urethane {Ethyl carbamate}
7440622	Vanadium (fume or dust)
1314621	Vanadium pentoxide
108054	Vinyl acetate
593602	Vinyl bromide
75014	Vinyl chloride
100403	4-Vinylcyclohexene
75025	Vinyl fluoride
75354	Vinylidene chloride
1206	Wood preservatives (containing arsenic and chromate)
1330207	Xylenes (mixed) including:
108383	m-Xylene
95476	o-Xylene
106423	p-Xylene
7440666	Zinc
*	Zinc compounds including but not limited to:
1314132	Zinc oxide

Appendix B

Health and Safety Code Related to Air Toxics Hot Spots, SB-352, and SB-25

Appendix B. Health and Safety Code Related to Hot Spots Program¹

PART 6. AIR TOXICS "HOT SPOTS" INFORMATION AND ASSESSMENT

(Part 6 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Note: Sections 44380 and 44384 became operative Jan. 1, 1988.)

CHAPTER 1. LEGISLATIVE FINDINGS AND DEFINITIONS

(Chapter 1 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44300. This part shall be known and may be cited as the Air Toxics "Hot Spots" Information and Assessment Act of 1987. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44301. The Legislature finds and declares all of the following:

- (a) In the wake of recent publicity surrounding planned and unplanned releases of toxic chemicals into the atmosphere, the public has become increasingly concerned about toxics in the air.
- (b) The Congressional Research Service of the Library of Congress has concluded that 75 percent of the United States population lives in proximity to at least one facility that manufactures chemicals. An incomplete 1985 survey of large chemical companies conducted by the Congressional Research Service documented that nearly every chemical plant studied routinely releases into the surrounding air significant levels of substances proven to be or potentially hazardous to public health.
- (c) Generalized emissions inventories compiled by air pollution control districts and air quality management districts in California confirm the findings of the Congressional Research Service survey as well as reveal that many other facilities and businesses which do not actually manufacture chemicals do use hazardous substances in sufficient quantities to expose, or in a manner that exposes, surrounding populations to toxic air releases.
- (d) These releases may create localized concentrations or air toxics "hot spots" where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including, but not limited to, cancer and contribute to the cumulative health risks of emissions from other sources in the area. In some cases where large populations may not be significantly affected by adverse health risks, individuals may be exposed to significant risks.

¹ AB564 Passed in the 1996 legislative session. The text will be added when the code is revised.

- (e) Little data is currently available to accurately assess the amounts, types, and health impacts of routine toxic chemical releases into the air. As a result, there exists significant uncertainty about the amounts of potentially hazardous air pollutants which are released, the location of those releases, and the concentrations to which the public is exposed.
- (f) The State of California has begun to implement a long-term program to identify, assess, and control ambient levels of hazardous air pollutants, but additional legislation is needed to provide for the collection and evaluation of information concerning the amounts, exposures, and short- and long-term health effects of hazardous substances regularly released to the surrounding atmosphere from specific sources of hazardous releases.
- (g) In order to more effectively implement control strategies for those materials posing an unacceptable risk to the public health, additional information on the sources of potentially hazardous air pollutants is necessary.
- (h) It is in the public interest to ascertain and measure the amounts and types of hazardous releases and potentially hazardous releases from specific sources that may be exposing people to those releases, and to assess the health risks to those who are exposed. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44302. The definitions set forth in this chapter govern the construction of this part. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44303. "Air release" or "release" means any activity that may cause the issuance of air contaminants, including the actual or potential spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of a substance into the ambient air and that results from the routine operation of a facility or that is predictable, including, but not limited to, continuous and intermittent releases and predictable process upsets or leaks. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44304. "Facility" means every structure, appurtenance, installation, and improvement on land which is associated with a source of air releases or potential air releases of a hazardous material. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44306. "Health risk assessment" means a detailed comprehensive analysis prepared pursuant to Section 44361 to evaluate and predict the dispersion of hazardous substances in the environment and the potential for exposure of human populations and to assess and quantify both the individual and population wide health risks associated with those levels of exposure. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44307. "Operator" means the person who owns or operates a facility or part of a facility. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44308. "Plan" means the emissions inventory plan which meets the conditions specified in Section

44342. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44309. "Report" means the emissions inventory report specified in Section 44341. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 2. FACILITIES SUBJECT TO THIS PART

(Chapter 2 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44320. This part applies to the following:

- (a) Any facility which manufactures, formulates, uses, or releases any of the substances listed pursuant to Section 44321 or any other substance which reacts to form a substance listed in Section 44321 and which releases or has the potential to release total organic gases, particulates, or oxides of nitrogen or sulfur in the amounts specified in Section 44322.
- (b) Except as provided in Section 44323, any facility which is listed in any current toxics use or toxics air emission survey, inventory, or report released or compiled by a district. A district may, with the concurrence of the state board, waive the application of this part pursuant to this subdivision for any facility which the district determines will not release any substance listed pursuant to Section 44321 due to a shutdown or a process change. (Amended by Stats. 1989, Ch. 1254, Sec. 7). References at the time of publication (see page iii): Regulations: 17, CCR, sections 90700-90703, 90704, 93303, 93306

44321. For the purposes of Section 44320, the state board shall compile and maintain a list of substances that contains, but is not limited to, all of the following:

- (a) Substances identified by reference in paragraph (1) of subdivision (b) of Section 6382 of the Labor Code and substances placed on the list prepared by the National Toxicology Program issued by the United States Secretary of Health and Human Services pursuant to paragraph (4) of Section 262 of Public Law 95-622 of 1978. For the purposes of this subdivision, the state board may remove from the list any substance which meets both of the following criteria:
 - (1) No evidence exists that it has been detected in air.
 - (2) The substance is not manufactured or used in California, or, if manufactured or used in California, because of the physical or chemical characteristics of the substance or the manner in which it is manufactured or used, there is no possibility that it will become airborne.
- (b) Carcinogens and reproductive toxins referenced in or compiled pursuant to Section

25249.8, except those which meet both of the criteria identified in subdivision (a).

- (c) The candidate list of potential toxic air contaminants and the list of designated toxic air contaminants prepared by the state board pursuant to Article 2 (commencing with

Section 39660) of Chapter 3.5 of Part 2, including, but not limited to, all substances currently under review and scheduled or nominated for review and substances identified and listed for which health effects information is limited.

- (d) Substances for which an information or hazard alert has been issued by the repository of current data established pursuant to Section 147.2 of the Labor Code.
- (e) Substances reviewed, under review, or scheduled for review as air toxics or potential air toxics by the Office of Air Quality Planning and Standards of the Environmental Protection Agency, including substances evaluated in all of the following categories or their equivalent: preliminary health and source screening, detailed assessment, intent to list, decision not to regulate, listed, standard proposed, and standard promulgated.
- (f) Any additional substances recognized by the state board as presenting a chronic or acute threat to public health when present in the ambient air, including, but not limited to, any neurotoxins or chronic respiratory toxins not included within subdivision (a), (b), (c), (d), or (e). (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44322. This part applies to facilities specified in subdivision (a) of Section 44320 in accordance with the following schedule:

- (a) For those facilities that release, or have the potential to release, 25 tons per year or greater of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective on July 1, 1988.
- (b) For those facilities that release, or have the potential to release, more than 10 but less than 25 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective July 1, 1989.
- (c) For those facilities that release, or have the potential to release, less than 10 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, the state board shall, on or before July 1, 1990, prepare and submit a report to the Legislature identifying the classes of those facilities to be included in this part and specifying a timetable for their inclusion. (Amended by Stats. 1989, Ch. 1254, Sec. 8.)

44323. A district may prepare an industrywide emissions inventory and health risk assessment for facilities specified in subdivision (b) of Section 44320 and subdivisions (a) and (b) of Section

44322, and shall prepare an industrywide emissions inventory for the facilities specified in subdivision (c) of Section 44322, in compliance with this part for any class of facilities that the district finds and determines meets all of the following conditions:

- (a) All facilities in the class fall within one four-digit Standard Industrial Classification Code.
- (b) Individual compliance with this part would impose severe economic hardships on the majority of the facilities within the class.
- (c) The majority of the class is composed of small businesses.
- (d) Releases from individual facilities in the class can easily and generically be characterized and calculated. (Amended by Stats. 1989, Ch. 1254, Sec. 9.)

44324. This part does not apply to any facility where economic poisons are employed in their pesticidal use, unless that facility was subject to district permit requirements on or before August 1, 1987. As used in this section, "pesticidal use" does not include the manufacture or formulation of pesticides. (Added by Stats. 1981, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44325. Any solid waste disposal facility in compliance with Section 41805.5 is in compliance with the emissions inventory requirements of this part. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 3. AIR TOXICS EMISSION INVENTORIES

(Chapter 3 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44340. (a) The operator of each facility subject to this part shall prepare and submit to the district a proposed comprehensive emissions inventory plan in accordance with the criteria and guidelines adopted by the state board pursuant to Section 44342.
- (b) The proposed plan shall be submitted to the district on or before August 1, 1989, except that, for any facility to which subdivision (b) of Section 44322 applies, the proposed plan shall be submitted to the district on or before August 1, 1990. The district shall approve, modify, and approve as modified, or return for revision and resubmission, the plan within 120 days of receipt.
- (c) The district shall not approve a plan unless all of the following conditions are met:
- (1) The plan meets the requirements established by the state board pursuant to Section 44342.
 - (2) The plan is designed to produce, from the list compiled and maintained pursuant to Section 44321, a comprehensive characterization of the full range of hazardous materials that are released, or that may be released, to the surrounding air from the facility. Air release data shall be collected at, or calculated for, the primary locations of actual and potential release for each hazardous material. Data shall be collected or calculated for all continuous, intermittent, and predictable air releases.
 - (3) The measurement technologies and estimation methods proposed provide state-of-the-art effectiveness and are sufficient to produce a true representation of the types and quantities of air releases from the facility.
 - (4) Source testing or other measurement techniques are employed wherever necessary to verify emission estimates, as determined by the state board and to the extent technologically feasible. All testing devices shall be appropriately located, as determined by the state board.
 - (5) Data are collected or calculated for the relevant exposure rate or rates of each hazardous material according to its characteristic toxicity and for the emission rate necessary to ensure a characterization of risk associated with exposure to releases of the hazardous material that meets the requirements of Section 44361. The source of all emissions shall be displayed or described. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44341. Within 180 days after approval of a plan by the district, the operator shall implement the plan and prepare and submit a report to the district in accordance with the plan. The district shall transmit all monitoring data contained in the approved report to the state board. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44342. The state board shall, on or before May 1, 1989, in consultation with the districts, develop criteria and guidelines for site-specific air toxics emissions inventory plans which shall be designed to comply with the conditions specified in Section 44340 and which shall include at least all of the following:

- (a) For each class of facility, a designation of the hazardous materials for which emissions are to be quantified and an identification of the likely source types within that class of facility. The hazardous materials for quantification shall be chosen from among, and may include all or part of, the list specified in Section 44321.
- (b) Requirements for a facility diagram identifying each actual or potential discrete emission point and the general locations where fugitive emissions may occur. The facility diagram shall include any nonpermitted and nonprocess sources of emissions and shall provide the necessary data to identify emission characteristics. An existing facility diagram which meets the requirements of this section may be submitted.
- (c) Requirements for source testing and measurement. The guidelines may specify appropriate uses of estimation techniques including, but not limited to, emissions factors, modeling, mass balance analysis, and projections, except that source testing shall be required wherever necessary to verify emission estimates to the extent technologically feasible. The guidelines shall specify conditions and locations where source testing, fence-line monitoring, or other measurement techniques are to be required and the frequency of that testing and measurement.
- (d) Appropriate testing methods, equipment, and procedures, including quality assurance criteria.
- (e) Specifications for acceptable emissions factors, including, but not limited to, those which are acceptable for substantially similar facilities or equipment, and specification of procedures for other estimation techniques and for the appropriate use of available data.
- (f) Specification of the reporting period required for each hazardous material for which emissions will be inventoried.
- (g) Specifications for the collection of useful data to identify toxic air contaminants pursuant to Article 2 (commencing with Section 39660) of Chapter 3.5 of Part 2.
- (h) Standardized format for preparation of reports and presentation of data.
- (i) A program to coordinate and eliminate any possible overlap between the requirements of this chapter and the requirements of Section 313 of the Superfund Amendment and Reauthorization Act of 1986 (Public Law 99-499). The state board shall design the guidelines and criteria to ensure that, in collecting data to be used for emissions inventories, actual measurement is utilized whenever necessary to verify the accuracy of emission estimates, to the extent technologically feasible. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44343. The district shall review the reports submitted pursuant to Section 44341 and shall, within 90 days, review each report, obtain corrections and clarifications of the data, and notify the Office of Environmental Health Hazard Assessment, the Department of Industrial Relations, and the city or county health department of its findings and determinations as a result of its review of the report. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Amended by Governor's Reorganization Plan No. 1 of 1991, §142.)

44344. Except as provided in Section 44391, emissions inventories developed pursuant to this chapter shall be updated every four years, in accordance with the procedures established by the state board. Those updates shall take into consideration improvements in measurement techniques and advancing knowledge concerning the types and toxicity of hazardous material released or potentially released. (Amended by Stats. 1993, Ch. 1041, Sec. 1. Effective January 1, 1994.)

44344.3.

- (a) A facility shall be granted an exemption by a district from further compliance with this part after meeting all of the following criteria:
 - (1) The facility was required to comply with this part only as a result of its particulate matter emissions.
 - (2) The facility has participated in, utilized data derived from, or is eligible to utilize data derived from, approved pooled source testing.
 - (3) The facility has submitted an emissions inventory plan and report that was subsequently accepted and approved.
 - (4) The facility has been designated by the district as a low priority facility under the guidelines set forth pursuant to this part for facility prioritization, and facility emissions do not present a significant health risk as specified in subdivision (b) of Section 44362.
 - (5) The facility handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock. (b) Subdivision (a) does not apply to a facility that, because of information provided pursuant to Section 44344.7, is reclassified as an intermediate or high priority facility by the district.
- (c) The operator of a facility that has been granted an exemption pursuant to this section shall biennially submit a statement to the district for the district's review, with a copy of the most recent emissions inventory for the facility, indicating that, except as to matters for which an emissions inventory update has been or will be submitted pursuant to Section 44344.7, there has been no significant change in facility operations or activities. The district shall not impose any fee upon the operator with regard to the submission of the statement. (Added by Stats. 1993, Ch. 1037, Sec. 1. Effective January 1, 1994.)

44344.5. The operator of any new facility that previously has not been subject to this part shall prepare and submit an emissions inventory plan and report. (Added by Stats. 1993, Ch. 1037, Sec. 2. Effective January 1, 1994.)

44344.7. The operator of a facility exempted pursuant to subdivision (a) of Section 44344.3 shall submit an emissions inventory update for those sources and substances for which a change in activities or operations has occurred, as follows:

- (a) The facility emits a newly listed substance.
- (b) A sensitive receptor has been established or constructed on or after January 1, 1994, within 500 meters of the facility.
- (c) The facility emits a substance for which the potency factor has increased.
- (d) The facility has begun emission of a listed substance not included in the previous emissions inventory. (Added by Stats. 1993, Ch. 1037, Sec. 3. Effective January 1, 1994.)

44345. (a) On or before July 1, 1989, the state board shall develop a program to compile and make available to other state and local public agencies and the public all data collected pursuant to this chapter.

(b) In addition, the state board, on or before March 1, 1990, shall compile, by district, emissions inventory data for mobile sources and area sources not subject to district permit requirements, and data on natural source emissions, and shall incorporate these data into data compiled and released pursuant to this chapter. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44346. (a) If an operator believes that any information required in the facility diagram specified pursuant to subdivision (b) of Section 44342 involves the release of a trade secret, the operator shall nevertheless make the disclosure to the district, and shall notify the district in writing of that belief in the report.

(b) Subject to this section, the district shall protect from disclosure any trade secret designated as such by the operator, if that trade secret is not a public record.

(c) Upon receipt of a request for the release of information to the public which includes information which the operator has notified the district is a trade secret and which is not a public record, the following procedure applies:

- (1) The district shall notify the operator of the request in writing by certified mail, return receipt requested.
- (2) The district shall release the information to the public, but not earlier than 30 days after the date of mailing the notice of the request for information, unless, prior to the expiration of the 30-day period, the operator obtains an action in an appropriate court for a declaratory judgment that the information is subject to protection under this section or for a preliminary injunction prohibiting disclosure of the information to the public and promptly notifies the district of that action.

(d) This section does not permit an operator to refuse to disclose the information required pursuant to this part to the district.

(e) Any information determined by a court to be a trade secret, and not a public record pursuant to this section, shall not be disclosed to anyone except an officer or employee of the district, the state, or the United States, in connection with the official duties of that officer or employee under any law for the protection of health, or to contractors with the district or the state and its employees if, in the opinion of the

district or the state, disclosure is necessary and required for the satisfactory performance of a contract, for performance of work, or to protect the health and safety of the employees of the contractor.

- (f) Any officer or employee of the district or former officer or employee who, by virtue of that employment or official position, has possession of, or has access to, any trade secret subject to this section, and who, knowing that disclosure of the information to the general public is prohibited by this section, knowingly and willfully discloses the information in any manner to any person not entitled to receive it is guilty of a misdemeanor. Any contractor of the district and any employee of the contractor, who has been furnished information as authorized by this section, shall be considered an employee of the district for purposes of this section.
- (g) Information certified by appropriate officials of the United States as necessary to be kept secret for national defense purposes shall be accorded the full protections against disclosure as specified by those officials or in accordance with the laws of the United States
- (g) As used in this section, "trade secret" and "public record" have the meanings and protections given to them by Section 6254.7 of the Government Code and Section 1060 of the Evidence Code. All information collected pursuant to this chapter, except for data used to calculate emissions data required in the facility diagram, shall be considered "air pollution emission data," for the purposes of this section. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 4. RISK ASSESSMENT

(Chapter 4 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44360. (a) Within 90 days of completion of the review of all emissions inventory data for facilities specified in subdivision (a) of Section 44322, but not later than December 1, 1990, the district shall, based on examination of the emissions inventory data and in consultation with the state board and the State Department of Health Services, prioritize and then categorize those facilities for the purposes of health risk assessment. The district shall designate high, intermediate, and low priority categories and shall include each facility within the appropriate category based on its individual priority. In establishing priorities pursuant to this section, the district shall consider the potency, toxicity, quantity, and volume of hazardous materials released from the facility, the proximity of the facility to potential receptors, including, but not limited to, hospitals, schools, day care centers, worksites, and residences, and any other factors that the district finds and determines may indicate that the facility may pose a significant risk to receptors. The district shall hold a public hearing prior to the final establishment of priorities and categories pursuant to this section.
- (b) (1) Within 150 days of the designation of priorities and categories pursuant to subdivision (a), the operator of every facility that has been included within the highest priority category shall prepare and submit to the district a health risk assessment pursuant to Section 44361. The district may, at its discretion, grant a 30-day extension for submittal of the health risk assessment.

- (2) Health risk assessments required by this chapter shall be prepared in accordance with guidelines established by the Office of Environmental Health Hazard Assessment. The office shall prepare draft guidelines which shall be circulated to the public and the regulated community and shall adopt risk assessment guidelines after consulting with the state board and the Risk Assessment Committee of the California Air Pollution Control Officers Association and after conducting at least two public workshops, one in the northern and one in the southern part of the state. The adoption of the guidelines is not subject to Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code. The scientific review panel established pursuant to Section 39670 shall evaluate the guidelines adopted under this paragraph and shall recommend changes and additional criteria to reflect new scientific data or empirical studies.
- (3) The guidelines established pursuant to paragraph (2) shall impose only those requirements on facilities subject to this subdivision that are necessary to ensure that a required risk assessment is accurate and complete and shall specify the type of site-specific factors that districts may take into account in determining when a single health risk assessment may be allowed under subdivision (d). The guidelines shall, in addition, allow the operator of a facility, at the operator's option, and to the extent that valid and reliable data are available, to include for consideration by the district in the health risk assessment any or all of the following supplemental information:
 - (a) Information concerning the scientific basis for selecting risk parameter values that are different than those required by the guidelines and the likelihood distributions that result when alternative values are used.
 - (b) Data from dispersion models, microenvironment characteristics, and population distributions that may be used to estimate maximum actual exposure.
 - (c) Risk expressions that show the likelihood that any given risk estimate is the correct risk value.
 - (d) A description of the incremental reductions in risk that occur when exposure is reduced.
- (4) To ensure consistency in the use of the supplemental information authorized by subparagraphs (A), (B), (C), and (D) of paragraph (3), the guidelines established pursuant to paragraph (2) shall include guidance for use by the districts in considering the supplemental information when it is included in the health risk assessment. (c) Upon submission of emissions inventory data for facilities specified in subdivisions (b) and (c) of Section 44322, the district shall designate facilities for inclusion within the highest priority category, as appropriate, and any facility so designated shall be subject to subdivision (b). In addition, the district may require the operator of any facility to prepare and submit health risk assessments, in accordance with the priorities developed pursuant to subdivision (a).
- (e) The district shall, except where site specific factors may affect the results, allow the use of a single health risk assessment for two or more substantially identical facilities operated by the same person.

- (f) Nothing contained in this section, Section 44380.5, or Chapter 6 (commencing with Section 44390) shall be interpreted as requiring a facility operator to prepare a new or revised health risk assessment using the guidelines established pursuant to paragraph (2) of subdivision (a) of this section if the facility operator is required by the district to begin the preparation of a health risk assessment before those guidelines are established. (Amended by Stats. 1992, Ch. 1162, Sec. 1. Effective January 1, 1993.)
44361. (a) Each health risk assessment shall be submitted to the district. The district shall make the health risk assessment available for public review, upon request. After preliminary review of the emissions impact and modeling data, the district shall submit the health risk assessment to the Office of Environmental Health Hazard Assessment for review and, within 180 days of receiving the health risk assessment, the office shall submit to the district its comments on the data and findings relating to health effects. The district shall consult with the state board as necessary to adequately evaluate the emissions impact and modeling data contained within the risk assessment.
- (b) For the purposes of complying with this section, the Office of Environmental Health Hazard Assessment may select a qualified independent contractor to review the data and findings relating to health effects. The office shall not select an independent contractor to review a specific health risk assessment who may have a conflict of interest with regard to the review of that health risk assessment. Any review by an independent contractor shall comply with the following requirements:
 - (1) Be performed in a manner consistent with guidelines provided by the office.
 - (2) Be reviewed by the office for accuracy and completeness.
 - (3) Be submitted by the office to the district in accordance with this section.
 - (c) The district shall reimburse the Office of Environmental Health Hazard Assessment or the qualified independent contractor designated by the office pursuant to subdivision (b), within 45 days of its request, for its actual costs incurred in reviewing a health risk assessment pursuant to this section.
 - (d) If a district requests the Office of Environmental Health Hazard Assessment to consult with the district concerning any requirement of this part, the district shall reimburse the office, within 45 days of its request, for the costs incurred in the consultation.
 - (e) Upon designation of the high priority facilities, as specified in subdivision (a) of Section 44360, the Office of Environmental Health Hazard Assessment shall evaluate the staffing requirements of this section and may submit recommendations to the Legislature, as appropriate, concerning the maximum number of health risk assessments to be reviewed each year pursuant to this section. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section
44384. Amended by Governor's Reorganization Plan No. 1 of 1991, §144.)
44362. (a) Taking the comments of the Office of Environmental Health Hazard Assessment into account, the district shall approve or return for revision and resubmission and then approve, the health risk assessment within 180 days of receipt. If the health risk

assessment has not been revised and resubmitted within 60 days of the district's request of the operator to do so, the district may modify the health risk assessment and approve it as modified.

- (b) Upon approval of the health risk assessment, the operator of the facility shall provide notice to all exposed persons regarding the results of the health risk assessment prepared pursuant to Section 44361 if, in the judgment of the district, the health risk assessment indicates there is a significant health risk associated with emissions from the facility. If notice is required under this subdivision, the notice shall include only information concerning significant health risks attributable to the specific facility for which the notice is required. Any notice shall be made in accordance with procedures specified by the district. (Added by Stats. 1981, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Amended by Governor's Reorganization Plan No. 1 of 1991, 145.)

44363. (a) Commencing July 1, 1991, each district shall prepare and publish an annual report which does all of the following:
- (1) Describes the priorities and categories designated pursuant to Section 44360 and summarizes the results and progress of the health risk assessment program undertaken pursuant to this part.
 - (2) Ranks and identifies facilities according to the degree of cancer risk posed both to individuals and to the exposed population.
 - (3) Identifies facilities which expose individuals or populations to any noncancer health risks.
 - (4) Describes the status of the development of control measures to reduce emissions of toxic air contaminants, if any.
- (b) The district shall disseminate the annual report to county boards of supervisors, city councils, and local health officers and the district board shall hold one or more public hearings to present the report and discuss its content and significance. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44364. The state board shall utilize the reports and assessments developed pursuant to this part for the purposes of identifying, establishing priorities for, and controlling toxic air contaminants pursuant to Chapter 3.5 (commencing with Section 39650) of Part 2. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44365. (a) If the state board finds and determines that a district's actions pursuant to this part do not meet the requirements of this part, the state board may exercise the authority of the district pursuant to this part to approve emissions inventory plans and require the preparation of health risk assessments.
- (b) This part does not prevent any district from establishing more stringent criteria and requirements than are specified in this part for approval of emissions inventories and requiring the preparation and submission of health risk assessments. Nothing in this part limits the authority of a district under any other provision of law to assess and regulate releases of hazardous substances. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44366. (a) In order to verify the accuracy of any information submitted by facilities pursuant to this part, a district or the state board may proceed in accordance with Section 41510. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 5. FEES AND REGULATIONS

(Chapter 5 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44380. (a) The state board shall adopt a regulation which does all of the following:
- (1) Sets forth the amount of revenue which the district must collect to recover the reasonable anticipated cost which will be incurred by the state board and the Office of Environmental Health Hazard Assessment to implement and administer this part.
 - (2) Requires each district to adopt a fee schedule which recovers the costs of the district and which assesses a fee upon the operator of every facility subject to this part. A district may request the state board to adopt a fee schedule for the district if the district's program costs are approved by the district board and transmitted to the state board by April 1 of the year in which the request is made.
 - (3) Requires any district that has an approved toxics emissions inventory compiled pursuant to this part by August 1 of the preceding year to adopt a fee schedule, as described in paragraph (2), which imposes on facility operators fees which are, to the maximum extent practicable, proportionate to the extent of the releases identified in the toxics emissions inventory and the level of priority assigned to that source by the district pursuant to Section 44360.
- (b) Commencing August 1, 1992, and annually thereafter, the state board shall review and may amend the fee regulation.
- (c) The district shall notify each person who is subject to the fee of the obligation to pay the fee. If a person fails to pay the fee within 60 days after receipt of this notice, the district, unless otherwise provided by district rules, shall require the person to pay an additional administrative civil penalty. The district shall fix the penalty at not more than 100 percent of the assessed fee, but in an amount sufficient in its determination, to pay the district's additional expenses incurred by the person's noncompliance. If a person fails to pay the fee within 120 days after receipt of this notice, the district may initiate permit revocation proceedings. If any permit is revoked, it shall be reinstated only upon full payment of the overdue fee plus any late penalty, and a reinstatement fee to cover administrative costs of reinstating the permit.
- (d) Each district shall collect the fees assessed pursuant to subdivision (a). After deducting the costs to the district to implement and administer this part, the district shall transmit the remainder to the Controller for deposit in the Air Toxics Inventory and Assessment Account, which is hereby created in the General Fund. The money in the account is available, upon appropriation by the Legislature, to the state board and the Office of Environmental Health Hazard Assessment for the purposes of

administering this part. (Amended by Stats. 1992, Ch. 375, Sec. 1. Effective January 1, 1993.)

44380.1. A facility shall be granted an exemption by a district from paying a fee in accordance with Section 44380 if all of the following criteria are met:

- (a) The facility primarily handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock.
- (b) The facility was required to comply with this part only as a result of its particulate matter emissions.
- (c) The fee schedule adopted by the district or the state board for these types of facilities is not solely based on toxic emissions weighted for potency or toxicity. (Added by Stats. 1993, Ch. 1037, Sec. 4. Effective January 1, 1994.)

44380.5. In addition to the fee assessed pursuant to Section 44380, a supplemental fee may be assessed by the district, the state board, or the Office of Environmental Health Hazard Assessment upon the operator of a facility that, at the operator's option, includes supplemental information authorized by paragraph (3) of subdivision (b) of Section 44360 in a health risk assessment, if the review of that supplemental information substantially increases the costs of reviewing the health risk assessment by the district, the state board, or the office. The supplemental fee shall be set by the state board in the regulation required by subdivision (a) of Section 44380 and shall be set in an amount sufficient to cover the direct costs to review the information supplied by an operator pursuant to paragraph (3) of subdivision (b) of Section 44360. (Added by Stats. 1992, Ch. 1162, Sec. 2. Effective January 1, 1993.)

44381. (a) Any person who fails to submit any information, reports, or statements required by this part, or who fails to comply with this part or with any permit, rule, regulation, or requirement issued or adopted pursuant to this part, is subject to a civil penalty of not less than five hundred dollars (\$500) or more than ten thousand dollars (\$10,000) for each day that the information, report, or statement is not submitted, or that the violation continues.
- (b) Any person who knowingly submits any false statement or representation in any application, report, statement, or other document filed, maintained, or used for the purposes of compliance with this part is subject to a civil penalty of not less than one thousand dollars (\$1,000) or more than twenty-five thousand dollars (\$25,000) per day for each day that the information remains uncorrected. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1988, pursuant to Section 44384.)

44382. Every district shall, by regulation, adopt the requirements of this part as a condition of every permit issued pursuant to Chapter 4 (commencing with Section 42300) of Part 4 for all new and modified facilities. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44384. Except for Section 44380 and this section, all provisions of this part shall become operative on July 1, 1988. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative January 1, 1988, by its own provisions.)

CHAPTER 6. FACILITY TOXIC AIR CONTAMINANT RISK REDUCTION AUDIT AND PLAN

(Chapter 6 added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44390. For purposes of this chapter, the following definitions apply:

- (a) "Airborne toxic risk reduction measure" or "ATRRM" means those in-plant changes in production processes or feedstocks that reduce or eliminate toxic air emissions subject to this part. ATRRM's may include:
 - (1) Feedstock modification.
 - (2) Product reformulations.
 - (3) Production system modifications.
 - (4) System enclosure, emissions control, capture, or conversion.
 - (5) Operational standards and practices modification.
- (b) Airborne toxic risk reduction measures do not include measures that will increase risk from exposure to the chemical in another media or that increase the risk to workers or consumers.
- (c) "Airborne toxic risk reduction audit and plan" or "audit and plan" means the audit and plan specified in Section 44392. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44391. (a) Whenever a health risk assessment approved pursuant to Chapter 4 (commencing with Section 44360) indicates, in the judgment of the district, that there is a significant risk associated with the emissions from a facility, the facility operator shall conduct an airborne toxic risk reduction audit and develop a plan to implement airborne toxic risk reduction measures that will result in the reduction of emissions from the facility to a level below the significant risk level within five years of the date the plan is submitted to the district. The facility operator shall implement measures set forth in the plan in accordance with this chapter.
- (b) The period to implement the plan required by subdivision (a) may be shortened by the district if it finds that it is technically feasible and economically practicable to implement the plan to reduce emissions below the significant risk level more quickly or if it finds that the emissions from the facility pose an unreasonable health risk.
 - (c) A district may lengthen the period to implement the plan required by subdivision (a) by up to an additional five years if it finds that a period longer than five years will not result in an unreasonable risk to public health and that requiring implementation of the plan within five years places an unreasonable economic burden on the facility operator or is not technically feasible.
 - (d)
 - (1) The state board and districts shall provide assistance to smaller businesses that have inadequate technical and financial resources for obtaining information, assessing risk reduction methods, and developing and applying risk reduction techniques.
 - (2) Risk reduction audits and plans for any industry subject to this chapter which is comprised mainly of small businesses using substantially similar technology may be completed by a self-conducted audit and checklist developed by the state

board. The state board, in coordination with the districts, shall provide a copy of the audit and checklist to small businesses within those industries to assist them to meet the requirements of this chapter.

- (e) The audit and plan shall contain all the information required by Section 44392.
- (f) The plan shall be submitted to the district, within six months of a district's determination of significant risk, for review of completeness. Operators of facilities that have been notified prior to January 1, 1993, that there is a significant risk associated with emissions from the facility shall submit the plan by July 1, 1993. The district's review of completeness shall include a substantive analysis of the emission reduction measures included in the plan, and the ability of those measures to achieve emission reduction goals as quickly as feasible as provided in subdivisions (a) and (b).
- (g) The district shall find the audit and plan to be satisfactory within three months if it meets the requirements of this chapter, including, but not limited to, subdivision (f). If the district determines that the audit and plan does not meet those requirements, the district shall remand the audit and plan to the facility specifying the deficiencies identified by the district. A facility operator shall submit a revised audit and plan addressing the deficiencies identified by the district within 90 days of receipt of a deficiency notice.
- (h) Progress on the emission reductions achieved by the plan shall be reported to the district in emissions inventory updates. Emissions inventory updates shall be prepared as required by the audit and plan found to be satisfactory by the district pursuant to subdivision (g).
- (i) If new information becomes available after the initial risk reduction audit and plan, on air toxics risks posed by a facility, or emission reduction technologies that may be used by a facility that would significantly impact risks to exposed persons, the district may require the plan to be updated and resubmitted to the district.
- (j) This section does not authorize the emission of a toxic air contaminant in violation of an airborne toxic control measure adopted pursuant to Chapter 3.5 (commencing with Section 39650) or in violation of Section 41700. (Amended by Stats. 1993, Ch. 1041, Sec. 2. Effective January 1, 1994.)

44392. A facility operator subject to this chapter shall conduct an airborne toxic risk reduction audit and develop a plan which shall include at a minimum all of the following:

- (a) The name and location of the facility.
- (b) The SIC code for the facility.
- (c) The chemical name and the generic classification of the chemical.
- (d) An evaluation of the ATRRM's available to the operator.
- (e) The specification of, and rationale for, the ATRRMs that will be implemented by the operator. The audit and plan shall document the rationale for rejecting ATRRMs that are identified as infeasible or too costly.
- (f) A schedule for implementing the ATRRMs. The schedule shall meet the time requirements of subdivision (a) of Section 44391 or the time period for implementing the plan set by the district pursuant to subdivision (b) or (c) of Section 44391, whichever is applicable.

- (g) The audit and plan shall be reviewed and certified as meeting this chapter by an engineer who is registered as a professional engineer pursuant to Section 6762 of the Business and Professions Code, by an individual who is responsible for the processes and operations of the site, or by an environmental assessor registered pursuant to Section 25570.3. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44393. The plan prepared pursuant to Section 44391 shall not be considered to be the equivalent of a pollution prevention program or a source reduction program, except insofar as the audit and plan elements are consistent with source reduction, as defined in Section 25244.14, or subsequent statutory definitions of pollution prevention. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44394. Any facility operator who does not submit a complete airborne toxic risk reduction audit and plan or fails to implement the measures set forth in the plan as set forth in this chapter is subject to the civil penalty specified in subdivision (a) of Section 44381, and any facility operator who, in connection with the audit or plan, knowingly submits any false statement or representation is subject to the civil penalty specified in subdivision (b) of Section 44381. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

SB-352

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BILL TEXT

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INTRODUCED BY Senator Escutia

FEBRUARY 19, 2003

An act to amend Section 17213 of the Education Code, and to amend Section 21151.8 of the Public Resources Code, relating to public schools.

LEGISLATIVE COUNSEL'S DIGEST

SB 352, Escutia. Schoolsites: sources of pollution.

Existing law sets forth various requirements regarding the siting, structural integrity, safety, and fitness-for-occupancy of school buildings, including, but not limited to, a prohibition of the approval by the governing board of a school district of the acquisition of a schoolsite by a school district, unless prescribed conditions relating to possible exposure to hazardous substances are satisfied, and a prohibition on the approval of a related environmental impact report or negative declaration.

This bill would, in addition, prohibit the approval by the governing board of a school district of a schoolsite that is within 500 feet from the edge of the closest traffic lane of a freeway or other busy traffic corridor, unless prescribed conditions are met and would make conforming and other technical, nonsubstantive changes.

Existing law requires the lead agency to consult with prescribed agencies to identify facilities that might reasonably be anticipated to emit hazardous materials, within 1/4 of a mile of the schoolsite.

This bill would define "facility" for this purpose and would require the lead agency to consult to identify freeways and other busy traffic corridors, as defined, large agricultural operations, and railyards, within 1/4 of a mile of the schoolsite, and would make conforming and other technical, nonsubstantive changes.

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1. The Legislature finds and declares all of the

following:

(a) Many studies have shown significantly increased levels of pollutants, particularly diesel particulates, in close proximity to freeways and other major diesel sources. A recent study of Los Angeles area freeways measured diesel particulate levels up to 25 times higher near freeways than those levels elsewhere. Much of the pollution from freeways is associated with acute health effects, exacerbating asthma and negatively impacting the ability of children to learn.

(b) Cars and trucks release at least forty different toxic air contaminants, including, but not limited to, diesel particulate, benzene, formaldehyde, 1,3-butadiene and acetaldehyde. Levels of these pollutants are generally concentrated within 500 feet of freeways and very busy roadways.

(c) Current state law governing the siting of schools does not specify whether busy freeways should be included in environmental impact reports of nearby "facilities." Over 150 schools are already estimated to be within 500 feet of extremely high traffic roadways.

(d) A disproportionate number of economically disadvantaged pupils may be attending schools that are close to busy roads, putting them at an increased risk of developing bronchitis from elevated levels of several pollutants associated with traffic. Many studies have confirmed that increased wheezing and bronchitis occurs among children living in high traffic areas.

(e) It is therefore the intent of the Legislature to protect school children from the health risks posed by pollution from heavy freeway traffic and other nonstationary sources in the same way that they are protected from industrial pollution.

SEC. 2. Section 17213 of the Education Code is amended to read:

17213. The governing board of a school district may not approve a project involving the acquisition of a schoolsite by a school district, unless all of the following occur:

(a) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, determines that the property purchased or to be built upon is not any of the following:

(1) The site of a current or former hazardous waste disposal site or solid waste disposal site, unless if the site was a former solid waste disposal site, the governing board of the school district concludes that the wastes have been removed.

(2) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(3) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood.

(b) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, in preparing the environmental impact report or negative declaration has consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and other busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous air emissions,

or to handle hazardous or acutely hazardous materials, substances, or waste. The school district, as the lead agency, shall include a list of the locations for which information is sought.

(c) The governing board of the school district makes one of the following written findings:

(1) Consultation identified none of the facilities or significant pollution sources specified in subdivision (b).

(2) The facilities or other pollution sources specified in subdivision (b) exist, but one of the following conditions applies:

(A) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the school.

(B) The governing board finds that corrective measures required under an existing order by another governmental entity that has jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes this finding, the governing board shall also make a subsequent finding, prior to the occupancy of the school, that the emissions have been mitigated to these levels.

(C) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(D) The governing board finds that neither of the conditions set forth in subparagraph (B) or (C) can be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(d) As used in this section:

(1) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located.

As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substance identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(2) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(3) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(4) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(5) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(6) "Administering agency" means any agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with

Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

SEC. 3. Section 21151.8 of the Public Resources Code is amended to read:

21151.8. (a) An environmental impact report or negative declaration may not be approved for any project involving the purchase of a schoolsite or the construction of a new elementary or secondary school by a school district unless all of the following occur:

(1) The environmental impact report or negative declaration includes information that is needed to determine if the property proposed to be purchased, or to be constructed upon, is any of the following:

(A) The site of a current or former hazardous waste disposal site or solid waste disposal site and, if so, whether the wastes have been removed.

(B) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(C) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood, or other nearby schools.

(D) A site that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor.

(2) The school district, as the lead agency, in preparing the environmental impact report or negative declaration has notified in writing and consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous emissions or handle hazardous or acutely hazardous materials, substances, or waste. The notification by the school district, as the lead agency, shall include a list of the locations for which information is sought.

(3) The governing board of the school district makes one of the following written findings:

(A) Consultation identified no facilities of this type or other significant pollution sources specified in paragraph (2).

(B) The facilities or other pollution sources specified in paragraph (2) exist, but one of the following conditions applies:

(i) The health risks from the facilities or other pollution

sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school.

(ii) Corrective measures required under an existing order by another agency having jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes a finding pursuant to this clause, it shall also make a subsequent finding, prior to occupancy of the school, that the emissions have been so mitigated.

(iii) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(C) The facilities or other pollution sources specified in paragraph (2) exist, but conditions in clause (i), (ii) or (iii) of subparagraph (B) cannot be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213 of the Education Code. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(4) Each administering agency, air pollution control district, or air quality management district receiving written notification from a lead agency to identify facilities pursuant to paragraph (2) shall provide the requested information and provide a written response to the lead agency within 30 days of receiving the notification. The environmental impact report or negative declaration shall be conclusively presumed to comply with this section as to the area of responsibility of any agency that does not respond within 30 days.

(b) If a school district, as a lead agency, has carried out the consultation required by paragraph (2) of subdivision (a), the environmental impact report or the negative declaration shall be conclusively presumed to comply with this section, notwithstanding any failure of the consultation to identify an existing facility or other pollution source specified in paragraph (2) of subdivision (a).

(c) As used in this section and Section 21151.4, the following definitions shall apply:

(1) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(2) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(3) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(4) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(5) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located.

As determined by the air pollution control officer, hazardous air

emissions also means emissions into the ambient air from any substances identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(6) "Administering agency" means an agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area, as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

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CHAPTER 731

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INTRODUCED BY Senator Escutia

(Principal coauthors: Assembly Members Kuehl and Villaraigosa)

(Coauthors: Senators Alarcon, Figueroa, Ortiz, Perata, Polanco, Sher, Solis, and Speier)

(Coauthors: Assembly Members Alquist, Aroner, Firebaugh, Honda, Jackson, Knox, Lempert, Mazzoni, Romero, Shelley, Steinberg, Thomson, Vincent, Washington, and Wildman)

DECEMBER 7, 1998

An act to amend Sections 39606, 39660, and 40451 of, to add Section 39617.5 to, to add Part 3 (commencing with Section 900) to Division 1 of, and to add Article 4.5 (commencing with Section 39669.5) to Chapter 3.5 of Part 2 of Division 26 of, the Health and Safety Code, relating to environmental health protection.

LEGISLATIVE COUNSEL'S DIGEST

SB 25, Escutia. Environmental health protection: children.

(1) Existing law requires the State Air Resources Board to adopt ambient air quality standards in consideration of specified factors, including public health effects, as provided, and to specify threshold levels for health effects in listing substances determined to be toxic air contaminants. Existing law requires the Office of Environmental Health Hazard Assessment, upon request of the state board, to evaluate the health effects of and prepare recommendations regarding specified substances which may be or are emitted into the

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ambient air and that may be determined to be toxic air contaminants.

Under existing law, the state board's request is required to be in accordance with an agreement that ensures that the office's workload in implementing these provisions will not be increased over that budgeted for the 1991-92 fiscal year, as provided.

This bill would eliminate the requirement for that agreement, and would impose specified requirements on the state board and the office generally relating to the protection of infants and children from environmental health hazards. The bill would require the state

board, not later than December 31, 2000, to review all existing health-based ambient air quality standards to determine whether the standards adequately protect the health of the public, including infants and children, and to revise the highest priority air quality standard determined to be inadequate, not later than December 31, 2002. The bill would require the office, by July 1, 2001, to establish a list of up to 5 specified toxic air contaminants that may cause infants and children to be especially susceptible to illness. The bill would require the state board to review and, as appropriate, revise any control measures adopted for those toxic air contaminants, to reduce exposure to those toxic air contaminants, as provided.

(2) Existing law requires the South Coast Air Quality Management District to notify all schools in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded.

This bill would also require the south coast district to notify day care centers in that basin, to the extent feasible and upon request. The bill would create a state-mandated local program by imposing new duties on the south coast district.

(3) The bill would create the Children's Environmental Health Center within the Environmental Protection Agency to, among other things, serve as chief advisor to the Secretary for Environmental Protection and to the Governor on matters within the jurisdiction of the agency relating to environmental health and environmental protection as it relates to children.

(4) This bill would incorporate additional changes to Section 40451 of the Health and Safety Code, proposed by SB 1195, to be operative only if SB 1195 and this bill are both chaptered on or before January 1, 2000, and this bill is chaptered last.

(5) The California Constitution requires the state to reimburse local agencies and school districts for certain costs mandated by the state. Statutory provisions establish procedures for making that reimbursement, including the creation of a State Mandates Claims Fund to pay the costs of mandates that do not exceed \$1,000,000 statewide and other procedures for claims whose statewide costs exceed \$1,000,000.

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This bill would provide that, if the Commission on State Mandates determines that the bill contains costs mandated by the state, reimbursement for those costs shall be made pursuant to these statutory provisions.

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1. The Legislature finds and declares all of the following:

(a) Infants and children have a higher ventilation rate than adults relative to their body weight and lung surface area, resulting

in a greater dose of pollution delivered to their lungs.

(b) Children have narrower airways than adults. Thus, irritation or inflammation caused by air pollution that would produce only a slight response in an adult can result in a potentially significant obstruction of the airway in a young child.

(c) Children spend significantly more time outdoors, especially in the summer, when ozone air pollution levels are typically highest. National statistics show that children spend an average of 50 percent more time outdoors than adults.

(d) Air pollution is known to exacerbate asthma and be a trigger for asthma attacks in infants and children, 500,000 of whom are afflicted with this chronic lung disease in California.

(e) Infant's and children's developing organs and tissues are more susceptible to damage from some environmental contaminants than are adult organs and tissues.

(f) It is the intent of the Legislature in enacting this act, to require that the state's air quality standards and airborne toxic control measures be reviewed to determine if they adequately protect the health of infants and children, and that these standards and measures be revised if they are determined to be inadequate.

(g) It is also the intent of the Legislature in enacting this act to require the State Air Resources Board and the Office of Environmental Health Hazard Assessment to consider the health impacts to all populations of children, including special subpopulations of infants and children that comprise a meaningful portion of the general population, such as children with asthma, cystic fibrosis, or other respiratory conditions or diseases, in setting or revising standards pursuant to this act.

SEC. 2. Part 3 (commencing with Section 900) is added to Division 1 of the Health and Safety Code, to read:

PART 3. CHILDREN'S ENVIRONMENTAL HEALTH CENTER

900. There is hereby created the Children's Environmental Health
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Center within the Environmental Protection Agency. The primary purposes of the center shall include all of the following:

(a) To serve as the chief advisor to the Secretary for Environmental Protection and to the Governor on matters within the jurisdiction of the Environmental Protection Agency relating to environmental health and environmental protection as each of those matters relates to children.

(b) To assist the boards, departments, and offices within the Environmental Protection Agency to assess the effectiveness of statutes, regulations, and programs designed to protect children from environmental hazards.

(c) To coordinate within the Environmental Protection Agency and with other state agencies, regulatory efforts, research and data collection, and other programs and services that impact the

environmental health of children, and coordinate with appropriate federal agencies conducting related regulatory efforts and research and data collection.

(d) In consultation with the State Air Resources Board and the Office of Environmental Health Hazard Assessment, and notwithstanding Section 7550.5 of the Government Code, to report to the Legislature and the Governor no later than December 31, 2001, on the progress of the state board and the office toward implementing the act that added this part during the 1999-2000 Regular Session and to make recommendations for any statutory or regulatory changes that may be necessary to carry out the intent of that act to protect the public health, including infants and children, from air pollutants and toxic air contaminants.

SEC. 3. Section 39606 of the Health and Safety Code is amended to read:

39606. (a) The state board shall do both of the following:

(1) Based upon similar meteorological and geographic conditions and consideration for political boundary lines whenever practicable, divide the state into air basins to fulfill the purposes of this division.

(2) Adopt standards of ambient air quality for each air basin in consideration of the public health, safety, and welfare, including, but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy. These standards may vary from one air basin to another. Standards relating to health effects shall be based upon the recommendations of the Office of Environmental Health Hazard Assessment.

(b) In its recommendations for submission to the state board pursuant to paragraph (2) of subdivision (a), the Office of Environmental Health Hazard Assessment, to the extent that information is available, shall assess the following:

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(1) Exposure patterns, including, but not limited to, patterns determined by relevant data supplied by the state board, among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.

(2) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.

(3) The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.

(4) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(c) In assessing the factors specified in subdivision (b), the

office shall use current principles, practices, and methods used by public health professionals who are experienced practitioners in the field of human health effects assessment. The scientific basis or scientific portion of the method used by the office to assess the factors set forth in subdivision (b) shall be subject to peer review as described in Section 57004 or in a manner consistent with the peer review requirements of Section 57004. Any person may submit any information for consideration by the entity conducting the peer review, which may receive oral testimony.

(d) (1) No later than December 31, 2000, the state board in consultation with the office, shall review all existing health-based ambient air quality standards to determine whether, based on public health, scientific literature, and exposure pattern data, the standards adequately protect the health of the public, including infants and children, with an adequate margin of safety. The state board shall publish a report summarizing these findings.

(2) The state board shall revise the highest priority ambient air quality standard determined to be inadequate to protect infants and children with an adequate margin of safety, based on its report, no later than December 31, 2002. Following the revision of the highest priority standard, the state board shall revise any additional standards determined to be inadequate to protect infants and children with an adequate margin of safety, at the rate of at least one per year. The standards shall be established at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety.

(e) Nothing in this section shall restrict the authority of the state board to consider additional information in establishing ambient air quality standards or to adopt an ambient air quality standard designed to protect vulnerable populations other than infants and children.

SEC. 4. Section 39617.5 is added to the Health and Safety Code, to A-8

read:

39617.5. (a) Not later than January 1, 2003, the state board shall do all of the following:

(1) Evaluate the adequacy of the current monitoring network for its ability to gather the data necessary to determine the exposure of infants and children to air pollutants including criteria air pollutants and toxic air contaminants.

(2) Identify areas where the exposure of infants and children to air pollutants is not adequately measured by the current monitoring network.

(3) Recommend changes to improve air pollution monitoring networks and data collection to more accurately reflect the exposure of infants and children to air pollutants.

(b) In carrying out this section, the state board, in cooperation

with the districts, shall expand its existing monitoring program in six communities around the state in nonattainment areas, as selected by the state board, to include special monitoring of children's exposure to air pollutants and toxic contaminants. The expanded program shall include placing air pollution monitors near schools, day care centers, and outdoor recreational facilities that are in close proximity to, or downwind from, major industrial sources of air pollutants and toxic air contaminants, including, freeways and major traffic areas. The purpose of the air pollution monitors shall be to conduct sampling of air pollution levels affecting children. Monitoring may include the use of fixed, mobile, and other monitoring devices, as appropriate.

(c) The expanded monitoring program shall include the following:

(1) Monitoring during multiple seasons and at multiple locations within each community at schools, day care centers, recreational facilities, and other locations where children spend most of their time.

(2) A combination of upgrading existing fixed monitoring sites, establishing new fixed monitoring sites, and conducting indoor and outdoor sampling and personal exposure measurements in each community to provide the most comprehensive data possible on the levels of children's exposure to air pollutants and toxic air contaminants.

(d) Data collected from expanded air quality monitoring activities conducted pursuant to this section may be used for any purpose authorized by law, including, but not limited to, determinations as to whether an area has attained or has not attained the state and national ambient air quality standards, if the monitoring devices from which the data was collected meet the monitoring requirements specified in Section 58.14 of Title 40 of the Code of Federal Regulations for special purpose monitors, all other monitoring requirements of Part 58 of Title 40 of the Code of Federal Regulations, and all applicable requirements specified in regulations A-9

adopted by the state board.

SEC. 5. Section 39660 of the Health and Safety Code is amended to read:

39660. (a) Upon the request of the state board, the office, in consultation with and with the participation of the state board, shall evaluate the health effects of and prepare recommendations regarding substances, other than pesticides in their pesticidal use, which may be or are emitted into the ambient air of California and that may be determined to be toxic air contaminants.

(b) In conducting this evaluation, the office shall consider all available scientific data, including, but not limited to, relevant data provided by the state board, the State Department of Health Services, the Occupational Safety and Health Division of the Department of Industrial Relations, the Department of Pesticide

Regulation, international and federal health agencies, private industry, academic researchers, and public health and environmental organizations. The evaluation shall be performed using current principles, practices, and methods used by public health professionals who are experienced practitioners in the fields of epidemiology, human health effects assessment, risk assessment, and toxicity.

(c) (1) The evaluation shall assess the availability and quality of data on health effects, including potency, mode of action, and other relevant biological factors, of the substance, and shall, to the extent that information is available, assess all of the following:

(A) Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.

(B) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.

(C) The effects on infants and children of exposure to toxic air contaminants and other substances that have a common mechanism of toxicity.

(D) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(2) The evaluation shall also contain an estimate of the levels of exposure that may cause or contribute to adverse health effects. If it can be established that a threshold of adverse health effects exists, the estimate shall include both of the following factors:

(A) The exposure level below which no adverse health effects are anticipated.

(B) An ample margin of safety that accounts for the variable effects that heterogeneous human populations exposed to the substance under evaluation may experience, the uncertainties associated with A-10

the applicability of the data to human beings, and the completeness and quality of the information available on potential human exposure to the substance. In cases in which there is no threshold of significant adverse health effects, the office shall determine the range of risk to humans resulting from current or anticipated exposure to the substance.

(3) The scientific basis or scientific portion of the method used by the office to assess the factors set forth in this subdivision shall be reviewed in a manner consistent with this chapter by the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670). Any person may submit any information for consideration by the panel, which may receive oral testimony.

(d) The office shall submit its written evaluation and

recommendations to the state board within 90 days after receiving the request of the state board pursuant to subdivision (a). The office may, however, petition the state board for an extension of the deadline, not to exceed 30 days, setting forth its statement of the reasons that prevent the office from completing its evaluation and recommendations within 90 days. Upon receipt of a request for extension of, or noncompliance with, the deadline contained in this section, the state board shall immediately transmit to the Assembly Committee on Rules and the Senate Committee on Rules, for transmittal to the appropriate standing, select, or joint committee of the Legislature, a statement of reasons for extension of the deadline, along with copies of the office's statement of reasons that prevent it from completing its evaluation and recommendations in a timely manner.

(e) (1) The state board or a district may request, and any person shall provide, information on any substance that is or may be under evaluation and that is manufactured, distributed, emitted, or used by the person of whom the request is made, in order to carry out its responsibilities pursuant to this chapter. To the extent practical, the state board or a district may collect the information in aggregate form or in any other manner designed to protect trade secrets.

(2) Any person providing information pursuant to this subdivision may, at the time of submission, identify a portion of the information submitted to the state board or a district as a trade secret and shall support the claim of a trade secret, upon the written request of the state board or district board. Subject to Section 1060 of the Evidence Code, information supplied that is a trade secret, as specified in Section 6254.7 of the Government Code, and that is so marked at the time of submission, shall not be released to any member of the public. This section does not prohibit the exchange of properly designated trade secrets between public agencies when those

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trade secrets are relevant and necessary to the exercise of their jurisdiction if the public agencies exchanging those trade secrets preserve the protections afforded that information by this paragraph.

(3) Any information not identified as a trade secret shall be available to the public unless exempted from disclosure by other provisions of law. The fact that information is claimed to be a trade secret is public information. Upon receipt of a request for the release of information that has been claimed to be a trade secret, the state board or district shall immediately notify the person who submitted the information, and shall determine whether or not the information claimed to be a trade secret is to be released to the public. The state board or district board, as the case may be, shall make its determination within 60 days after receiving the request for disclosure, but not before 30 days following the

notification of the person who submitted the information. If the state board or district decides to make the information public, it shall provide the person who submitted the information 10 days' notice prior to public disclosure of the information.

(f) The office and the state board shall give priority to the evaluation and regulation of substances based on factors related to the risk of harm to public health, amount or potential amount of emissions, manner of, and exposure to, usage of the substance in California, persistence in the atmosphere, and ambient concentrations in the community. In determining the importance of these factors, the office and the state board shall consider all of the following information, to the extent that it is available:

(1) Research and monitoring data collected by the state board and the districts pursuant to Sections 39607, 39617.5, 39701, and 40715, and by the United States Environmental Protection Agency pursuant to paragraph (2) of subsection (k) of Section 112 of the federal act (42 U.S.C. Sec. 7412(k)(2)).

(2) Emissions inventory data reported for substances subject to Part 6 (commencing with Section 44300) and the risk assessments prepared for those substances.

(3) Toxic chemical release data reported to the state emergency response commission pursuant to Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986 (42 U.S.C. Sec. 11023) and Section 6607 of the Pollution Prevention Act of 1990 (42 U.S.C. Sec. 13106).

(4) Information on estimated actual exposures to substances based on geographic and demographic data and on data derived from analytical methods that measure the dispersion and concentrations of substances in ambient air.

SEC. 6. Article 4.5 (commencing with Section 39669.5) is added to Chapter 3.5 of Part 2 of Division 26 of the Health and Safety Code, A-12

to read:

Article 4.5. Special Provisions For Infants And Children

39669.5. The Legislature finds and declares that certain toxic air contaminants may pose risks that cause infants and children to be especially susceptible to illness and that certain actions are necessary to ensure their safety from toxic air contaminants.

(a) By July 1, 2001, the following shall occur:

(1) The office, in consultation with the state board, shall establish a list of up to five toxic air contaminants identified or designated by the state board pursuant to Section 39657 that may cause infants and children to be especially susceptible to illness. In developing the list, the office shall take into account public exposures to toxic air contaminants, whether by themselves or interacting with other toxic air contaminants or criteria pollutants, and the factors listed in subdivision (c) of Section 39660. The

office shall submit a report containing the list and its reasons for including the toxic air contaminants on the list to the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670).

(2) The scientific review panel, in a manner consistent with this chapter, shall review the list of toxic air contaminants submitted by the office pursuant to paragraph (1). As part of the review, any person may submit any information for consideration by the panel, which may receive oral testimony.

(b) (1) Within two years of the establishment of the list required pursuant to subdivision (a), the state board shall review and, as appropriate, revise any control measures adopted for the toxic air contaminants identified on the list, to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, and particularly infants and children.

(2) Within three years of the establishment of the list required pursuant to subdivision (a), for up to five of those toxic air contaminants for which no control measures have been previously adopted, the state board shall prepare a report on the need for regulations, following the procedure specified in Section 39665. The state board shall adopt within that same three-year timeframe, as appropriate, any new control measures to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, particularly infants and children.

(c) Beginning July 1, 2004, the office shall annually evaluate at least 15 toxic air contaminants identified or designated by the state board pursuant to Section 39657, and provide threshold exposure A-13

levels and nonthreshold health values, as appropriate, for those toxic air contaminants. The activities required pursuant to this subdivision shall continue until all toxic air contaminants are evaluated. The levels shall be established pursuant to the procedures adopted for health and risk assessments pursuant to paragraph (2) of subdivision (b) of Section 44360, and taking into account the factors listed in subdivision (c) of Section 39660.

Based on this evaluation, and after review by the scientific review panel as prescribed in paragraph (2) of subdivision (a), the office shall update the list established pursuant to subdivision (a), by July 1, 2005, and each year thereafter. Within three years of the initial or subsequent listing update, for up to five of the toxic air contaminants contained on that list for which no control measures have been previously adopted, or for at least five of the toxic air contaminants if more than five toxic air contaminants have been identified, the state board shall prepare a report on the need for regulation, following the procedure specified in Section 39665. The state board shall adopt within that three-year timeframe, as

appropriate, new control measures, pursuant to Article 4 (commencing with Section 39665), to reduce exposure to those toxic air contaminants, to protect public health, and particularly infants and children.

(d) Toxic air contaminants evaluated and listed pursuant to this section shall not include substances in those uses that are not subject to regulation by the state board pursuant to this chapter.

SEC. 7. Section 40451 of the Health and Safety Code is amended to read:

40451. (a) The south coast district shall use the Pollutant Standards Index developed by the Environmental Protection Agency and shall report and forecast pollutant levels daily for dissemination in the print and electronic media.

(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded.

(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards are exceeded.

(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded and the number of

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days and locations of these occurrences.

SEC. 7.5. Section 40451 of the Health and Safety Code is amended to read:

40451. (a) The south coast district shall use the Pollutant Standards Index developed by the United States Environmental Protection Agency and shall report and forecast pollutant levels daily for dissemination in the print and electronic media.

Commencing July 1, 2001, the south coast district shall also include in its report and forecast levels of PM_{2.5} in excess of the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.

(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded. Commencing July 1, 2001, using communication facilities available to it, the south coast district

shall also notify all schools in the South Coast Air Basin when the ambient level of PM_{2.5} is predicted to exceed the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.

(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards and the 24-hour federal ambient air standard for PM_{2.5}, as adopted in July 1997, or any standards adopted by the United States Environmental Protection Agency that succeed those standards, are exceeded.

(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded. Commencing July 1, 2001, the south coast district shall also include in that report the number of days and locations on and at which the 24-hour federal ambient air standard for PM_{2.5}, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard, is exceeded.

SEC. 8. Section 7.5 of this bill incorporates amendments to Section 40451 of the Health and Safety Code proposed by both this bill and SB 1195. It shall only become operative if

(1) both bills are enacted and become effective on or before January 1, 2000, (2) each bill amends Section A-15

40451 of the Health and Safety Code, and (3) this bill is enacted after SB 1195, in which case Section 7 of this bill shall not become operative.

SEC. 9. Notwithstanding Section 17610 of the Government Code, if the Commission on State Mandates determines that this act contains costs mandated by the state, reimbursement to local agencies and school districts for those costs shall be made pursuant to Part 7 (commencing with Section 17500) of Division 4 of Title 2 of the Government Code. If the statewide cost of the claim for reimbursement does not exceed one million dollars (\$1,000,000), reimbursement shall be made from the State Mandates Claims Fund.

Appendix C
Spatial Averaging of Receptors
for Toxics Risk Assessments

C.1 Summary

Air dispersion modeling for long term averages for risk assessments usually feature the single receptor with the highest concentration at the Point of Maximum Impact (PMI), at an existing residence, the residential maximally exposed individual (RMEI), at an existing offsite workplace, and the worker maximally exposed individual (WMEI). Individuals at a residence or a workplace will tend to move around and not remain at a single point. It therefore seemed reasonable to the ARB and OEHHA to compare modeled air concentrations at a single point with the air concentrations averaged over an area where exposure might more realistically occur. Appendix C compares modeled average air concentrations of several sized averaging domains with the estimate at the PMI. It also looks at area, volume, point and line sources to determine the impact of source type and size of source on the ratio of the PMI to averaged domain. The analysis presented in this document shows how the spatial average of the collective nearby receptors can be approximately 65% to 80% of the highest concentration. The spatial averaging of air concentrations at receptors is more sensitive to emissions from small sources versus large sources. The spatial averages for nearby areas as small as (10m x 10m) up to (100m x 100m) are shown.

C.2 Introduction

Since the inception of the “Hot Spots” and the air toxics programs in California, health risk assessment (HRA) results for an individual have typically been based on air dispersion modeling results at a single point or location. This method has been traditionally used for all types of receptors (e.g., Point of Maximum Impact, Maximum Exposed Individual Residence, Maximum Exposed Individual Worker, pathway receptors, etc.). The assumptions used in a risk assessment are designed to prevent underestimation of health impacts to the public – a health protective approach.

Air pollutant concentrations are estimated at receptors which are distributed in a grid pattern of sufficient size and density to capture the maximum concentration (e.g., at the Point of Maximum Impact (PMI)). Under some conditions, the PMI may be significantly higher than receptors only a few meters away. A more refined inhalation exposure estimate in such situations can be obtained by estimating an average concentration in a small area where the receptor might be moving about.

The Air Resources Board (ARB), in conjunction with the Office of Environmental Health Hazard Assessments (OEHHA), performed sensitivity analyses to evaluate the impacts of spatially averaging air dispersion modeling results. In this paper, we study the sensitivity of spatially averaging the concentration of a group of receptors in the vicinity of the PMI in order to obtain an average concentration that better represents the long-term average over space and time. That information is presented below.

C.3 Source Types

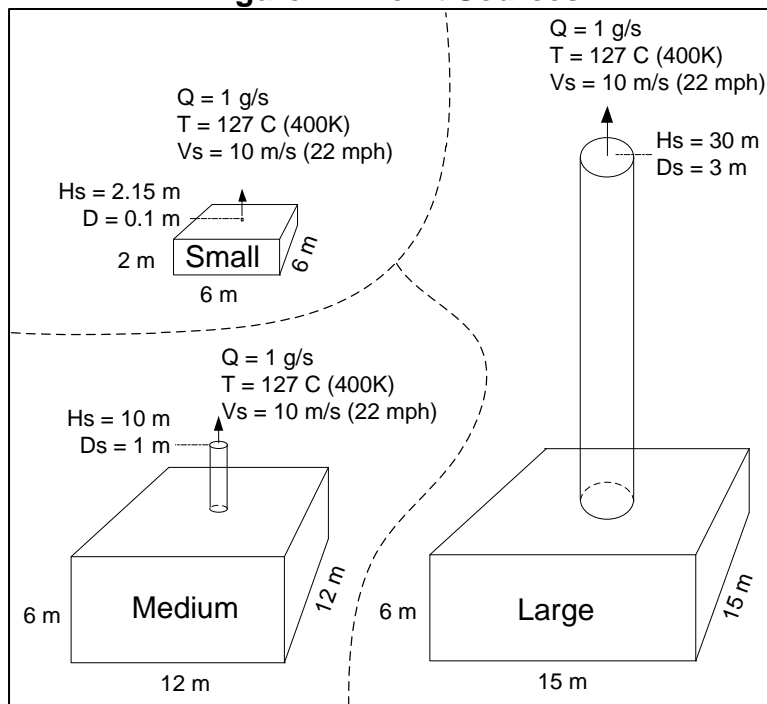
Air quality modeling of facility emissions are normally carried out with a Gaussian plume model such as US-EPA's AERMOD¹. The AERMOD algorithms include features that allow for the modeling of point, volume, and area sources. Line sources can be a special case of a series of volume or area sources.

For this analysis, we categorize each of the four source types (point, volume, area, and line) into three sizes; small, medium, and large. (Line sources are only treated as small and large.) The release parameters for input to the dispersion model are summarized in Tables 1, 2, 3, and 4. These sources are depicted schematically in Figures 1, 2, 3, and 4.

Air dispersion modeling for line sources is completed with the CAL3QHCR² model. CAL3QHCR is a roadway line source model. The line sources represented in this sensitivity analysis are roadway motor vehicle emissions. Roadways are not part of the Hot Spots program because the program only addresses stationary sources. However, roadways need to be modeled for proposed school sites within 500 feet of a busy roadway under SB-352. SB-352 specifies that the Hot Spots risk assessment guidance is used for the risk assessment. Differences between AERMOD and CAL3QHCR are beyond the scope of this paper. The concepts of spatial averaging with CAL3QHCR results could be extended to AERMOD line source studies.

¹ AERMOD – A steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain. U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model - AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

² CAL3QHCR – Line Source Model – Environmental Protection Agency, 1992. User's Guide for CAL3QHC Version 2: A Modeling Methodology for Predicting Pollutant Concentrations near Roadway Intersections. Publication No. EPA-454/R- 92-006. Office of Air Quality Planning & Standards, Research Triangle Park, NC. (NTIS No. PB 93-210250)

Figure 1 – Point Sources**Table 1 – Point Source (Stack) Modeling Parameters**

Source Size	Q_s (g/s)	H_s (m)	D_s (m)	$T_s^{(a)}$ (K)	V_s (m/s)	FPH ^(b) (m)	Bh (m)	Bl (m)	Xadj Yadj (m)	Similar Sources
Large	1	30	3	400	10	370.	6	15	7.5	Power Plant / Boiler
Medium	1	10	1	400	10	97.8	6	12	6	Asphalt Batch Plant
Small	1	2.15	0.1	400	10	5.15	2	6	3	Truck Engine

a) 400 K (260 F) is at the lower end of the combustion exhaust temperature range.
b) FPH (Final Plume Height) varies with atmospheric conditions and is calculated hourly by the air quality model. For this table we calculated the FPH with US-EPA's SCREEN3 model under neutral atmospheric stability (D) and low wind speed (1m/s) for comparative purposes.

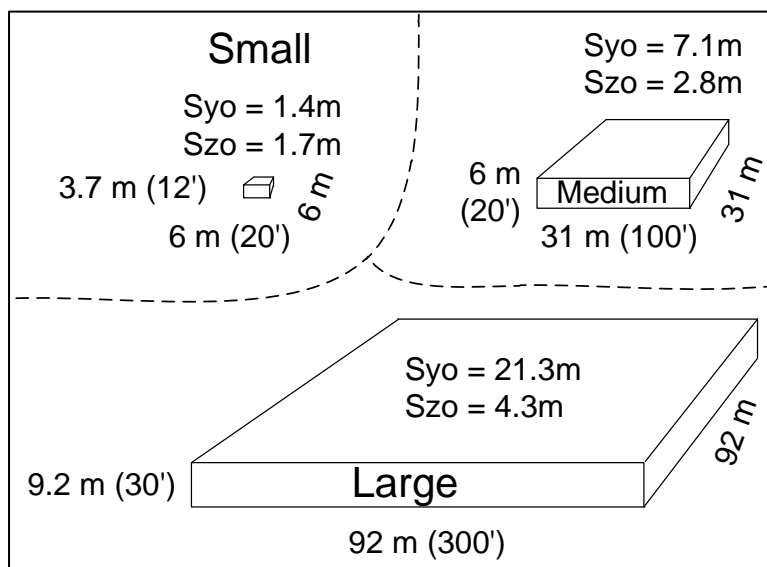
Figure 2 – Volume Sources

Table 2 – Volume Source Modeling Parameters					
Source Size	Qs (g/s)	Hs (m)	Syo (m)	Szo (m)	Similar Sources
Large	1	4.6	21.3 (L=92m)	4.3	Fleet Facility (300'x300'x30')
Medium	1	3.0	7.1 (L=31m)	2.8	(100'x100'x20')
Small	1	1.8	1.4 (L=6m)	1.7	Dry Cleaner (20'x20'x12')
H: Volume source height Hs: Plume centerline release height ($H = 2 H_s$) Syo: Initial plume dispersion in the horizontal ($S_{yo} = L / 4.3$) Szo: Initial plume dispersion in the vertical ($S_{zo} = H / 2.15$)					

Figure 3 – Area Sources

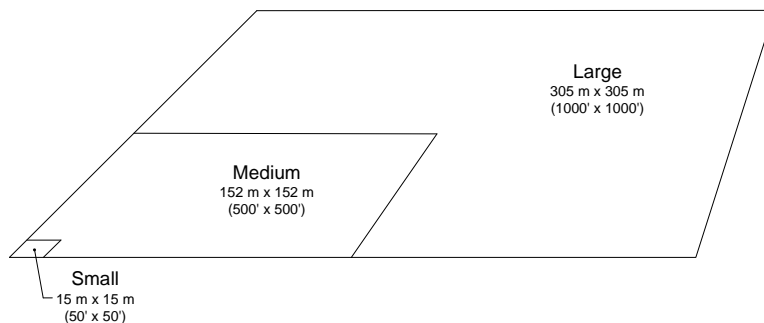
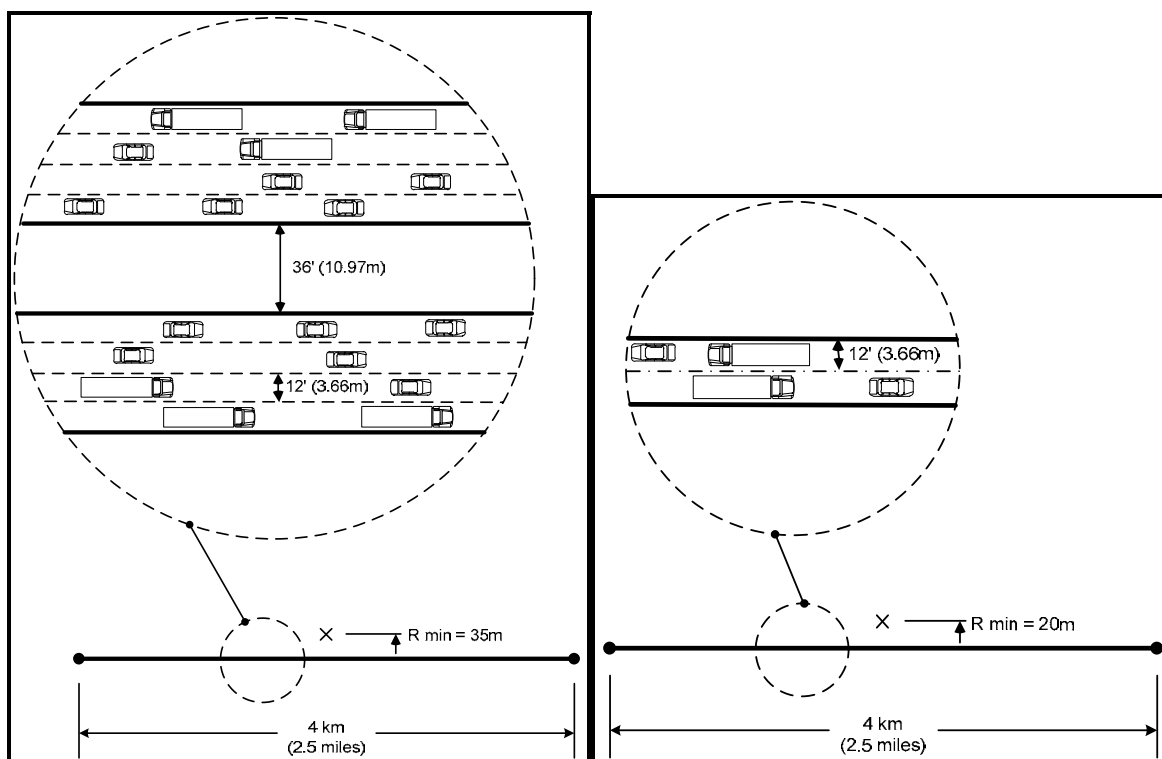


Table 3 – Area Source Modeling Parameters				
Source Size	Qs (g/s)	Hs (m)	Ls (m)	Similar Sources
Large	1	3.0	305	Rail Facility (1000'x1000')
Medium	1	3.0	152	Loading Industrial Facility (500'x500')
Small	1	2.0	15	Pile (50'x50')

Figure 4 – Line Source – Large and Small**Table 4 – Line Source Modeling Parameters**

Source Size	Qs (g/s)	Vehicles per Day	Lanes	Ls (m)	Min Receptor Placement (m)
Large	1	250,000	8	4000	35
Small	1	5,000	2	4000	20

The roadway line source is simulated as four kilometers of straight roadway. The large source is an eight lane roadway where the first receptor is located 35 m from the edge of the roadway. The small source is a two lane roadway where the first receptor is located 20 meters from the edge of the roadway. Hourly variations in traffic flow are shown in the Appendix 1.

US-EPA Guidelines³ accept the CALINE3 and CAL3QHCR models to simulate emissions from roadways. Algorithms to simulate the enhanced mechanical turbulence and thermal buoyancy associated with motor vehicles are included in the CALINE series of models. CALINE is formulated with the Pasquill-Gifford plume distributions to simulate downwind dispersion. AERMOD is US-EPA's state-of-science dispersion model. AERMOD does not use the Pasquill-Gifford step functions of dispersion curves for estimating atmospheric stability, but rather a continuum of atmospheric dispersion is

³ U.S. EPA (2005). Federal Register / Volume 70, Number 216 / November 9, 2005 / Rules and Regulations, 40 CFR Part 51 Appendix W, Revision to the Guideline on Air Quality Models, U.S. Environmental Protection Agency

simulated. However, AERMOD does not facilitate the hourly mechanical turbulence or thermal buoyancy associated with motor vehicles.

CAL3QHCR is used for the roadway motor vehicle emissions. Although there is potential to carefully apply AERMOD to line sources, comparing the results from these two models is beyond the scope of this sensitivity study.

C.4 Meteorological Data

AERMET is the computer program that processes and prepares meteorological data for use in AERMOD. Meteorological data that have been processed with the AERMET processor are obtained from various Districts. The latest consecutive years (up to five) were obtained. We selected the following stations for this analysis. Also see Figure 5.

- Costa Mesa (2005-2007)
- Fresno Air Terminal (2004-2008)
- Kearny Mesa (2003-2005)
- Lynwood (2005-2007)
- San Bernardino (2005-2007)

Figure 5 – Meteorological Station Locations



Wind rose summaries for each meteorological station are available in Appendix 2. The data for Costa Mesa, Lynwood, and San Bernardino are provided by the South Coast Air Quality Management District. Fresno Air Terminal (FAT) data are provided by the San Joaquin Valley Air Pollution Control District. Kearny Mesa data are provided by the San Diego Air Pollution Control District.

CAL3QHCR is a version of CALINE that can be used to simulate roadway emissions and also accepts a complete year of hourly meteorological data. CAL3QHCR requires meteorological data with Pasquill-Gifford (PG) classifications for stability. The meteorological data provided for AERMOD as discussed above does not include PG stability. Rather a continuum of stability is represented.

For the purpose of using CAL3QHCR in this sensitivity study, the PG stability class is estimated from the Monin-Obukhov length available in the AERMET processed meteorological data. As suggested by Sykes and Lewellen 1992⁴, the relationship between Monin-Obukhov length and PG stability class is shown in Table 5.

Table 5 – Stability Estimates	
PG Stability Class	Monin-Obukhov Length (m)
A	-5
B	-12.5
C	-50
D	-1000
E	25
F	13
As suggested by Sykes, R.I. and W.S. Lewellen (1992), "Review of potential models for UF ₆ dispersion," Martin Marietta Energy Systems, Inc., Safety and Analysis Report-19 (SAR-19)	

For regulatory purposes, we recommend that the stability class be determined with standard procedures for processing meteorological data with PG stability such as those available for the Industrial Source Complex – Short Term dispersion model.

The mixing height is constant at 500 meters for the CAL3QHCR simulations.

⁴ Sykes, R.I. and W.S. Lewellen (1992), "Review of potential models for UF₆ dispersion," Martin Marietta Energy Systems, Inc., Safety and Analysis Report-19 (SAR-19).

C.5 Receptors

Receptors are set as flagpoles 1.2 meters above ground. A coarse receptor grid with 20 meters spacing is used to locate and center a nested grid with five meter spacing on the point of maximum impact (PMI). We selected the PMI no closer than 20 meters to a point source; 20 meters to the virtual edge of a volume source; or zero meters to the edge of an area source. AERMOD limitations on receptor placement are that no receptors be located within one meter of the point source and no receptors within a volume source. Receptors within an area source are still valid.

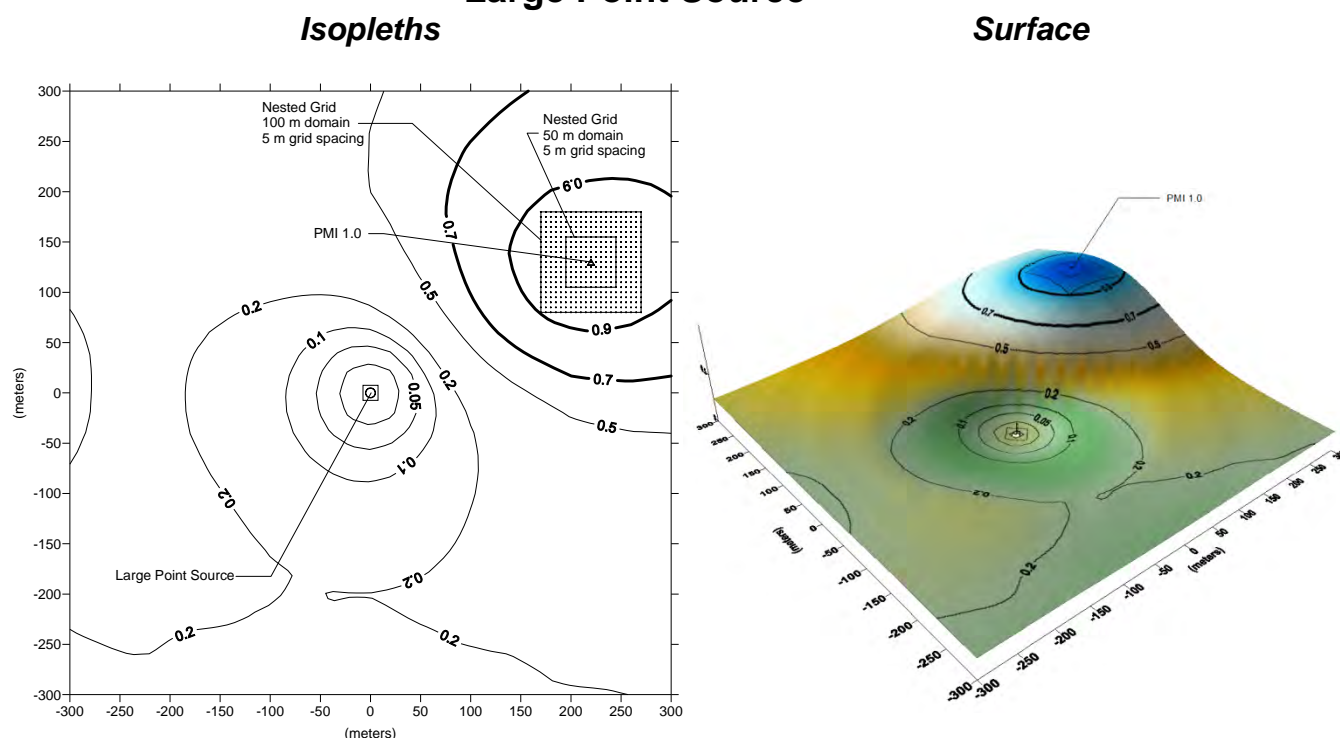
The nested grid was centered on the PMI for the large and medium point source receptors. For the small point source, volume sources, area sources, and line sources, the near edge of the grid was centered on the PMI in order to keep nested receptors off of the source. Simple arithmetic averaging was used to average the nested grid over the PMI with various nesting domain sizes. Figure 6 shows the PMI and two nested grids for the large point source.

Appendix 3 shows the PMI and two nested grids for each source (point, volume, area, and line) and for all sizes.

The spatial average was calculated for nested grids at ten different domains; 10m x 10m up to 100m x 100m, even though only two nested grids are shown on each plot.

An emission rate of 1 g/s was used for each source type. The resulting concentration field output was normalized to the offsite PMI. Therefore, the offsite receptor concentrations have a maximum value of 1.00 $\mu\text{g}/\text{m}^3$.

Figure 6
Concentration Distribution (Normalized to PMI)
Large Point Source



C.6 Results

The graphical displays of the concentration fields from the multitude of source types and meteorological representation are available in Appendix 3. It is evident from these figures that estimated ground level concentrations fall off most steeply from the PMI with smaller source types with a low plume rise where the PMI is located at the property fence line. This is to say that the spatial average is lowest relative to the PMI with this type of small source. Source types with high plume rise (e.g., tall stacks in Figures AP3.1.1 – 1.5) show a PMI far downwind where the concentration gradient is more gradual and therefore the difference between the estimated air concentration with the spatial average and the PMI is less..

The results of the spatial averaging are summarized in Figures 7 – 10. Supporting Tables are available in Appendix 4.

The spatial averaging for a 10m x 10m receptor field can be as low as 65% of the PMI value as seen in Table AP4.3.3 and Figure 9.3.

In addition, the graphical displays in Appendix 3 show that the dominate plume centerline is sometimes tilted from the cardinal directions. Since the nested grids for

spatial averaging were placed along the cardinal directions, the results in Appendix 4 may underestimate a spatial average centered on the dominate plume centerline. Appendix 5 shows how tilting the nested grid to coincide with the dominate plume centerline can increase the value of the spatial average. The value of the spatial averaged tilted grid may be higher than the non-tilted counterpart (e.g., 0.69 verses 0.59). Whether or not to tilt the grid is a subjective decision and should be considered on a case-by-case basis.

C.7 Recommendations

Spatial averaging may be used to estimate a long term concentration over a small nested grid of receptors to represent an area verses a single location as determined by the Point of Maximum Impact (PMI). Spatial averaging is most applicable for the following conditions.

- Long term averages are being calculated to represent multi-year impacts.
- The Point of Maximum Impact (PMI) is located at the fence line and close to the emission source.
- The concentration gradient is high near the PMI. This is more associated with low level plumes such as fugitive, volume, or area sources.

The following are recommendations for calculating the spatial average.

1. Spatial averaging should not be used for maximum one hour air concentration estimation.
2. Locate the off-site PMI with a nested grid resolution spacing of no greater than five meters. Two or more model runs with successively finer grid resolutions centered on the new PMI may be required to locate the final PMI.
3. Center the nested grid on the off-site receptors about the PMI. Limit the nested grid to 20m x 20m. The grid resolution spacing should be no greater than five meters. With a 5m grid resolution, the 20m x 20m nest will result in 25 receptors.
4. If necessary, tilt the nested grid to coincide with the dominate plume centerline. Polar receptors are easier to implement than a tilted rectangular grid. The domain of the polar receptor field should be limited to a 15 meter polar radius.

Although this sensitivity study evaluated nested grids up to 100m x 100m, the above recommendation is to limit the nested grid domain to 20m x 20m if rectangular and a radius of 15m if polar. (A 20m x 20m square area is equivalent to a 16m radius half circle. Therefore we rounded down to 15m radius for convenience.)

As a frame of reference, low density single family detached dwellings have been described in some city municipal codes as RD4 – RD7 zoning. RD4 allows four units per acre of land and RD7 allows seven units per acre of land. Table 6 shows the equivalent acreage and size in meters of RD4 – RD7 lots assuming uniformly distributed and square lots.

Table 6 – Residential Zoning vs Lot Size		
Zone	Lot Size (acres)	Lot Size Square Meter
RD4	0.250	32m x 32m
RD5	0.200	28m x 28m
RD7	0.143	24m x 24m
-	0.099	20m x 20m

Figure 7.1

Large Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

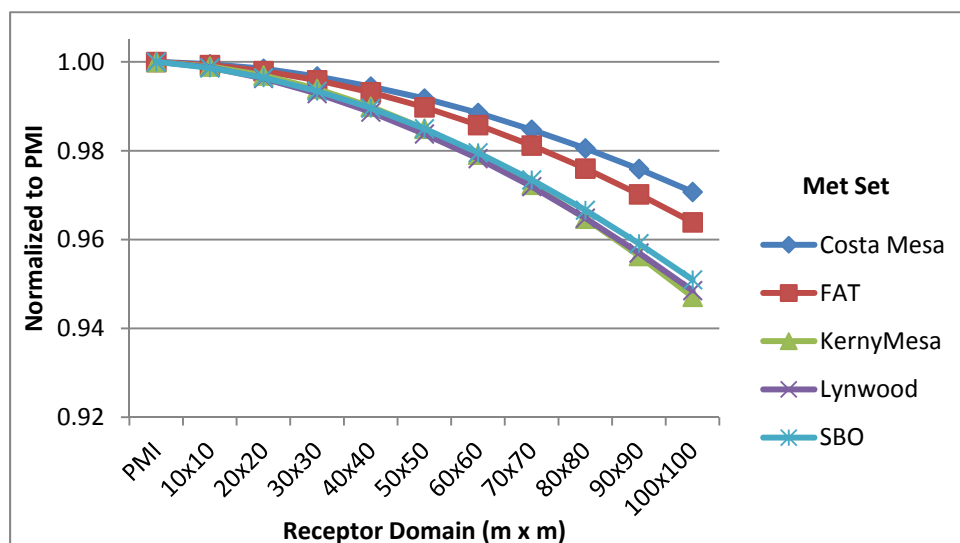


Figure 7.2

Medium Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

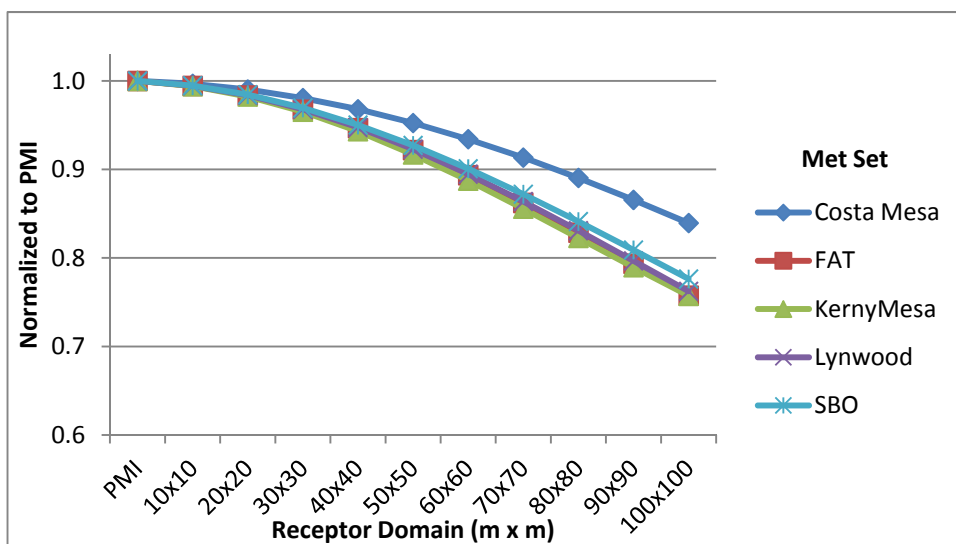


Figure 7.3

Small Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

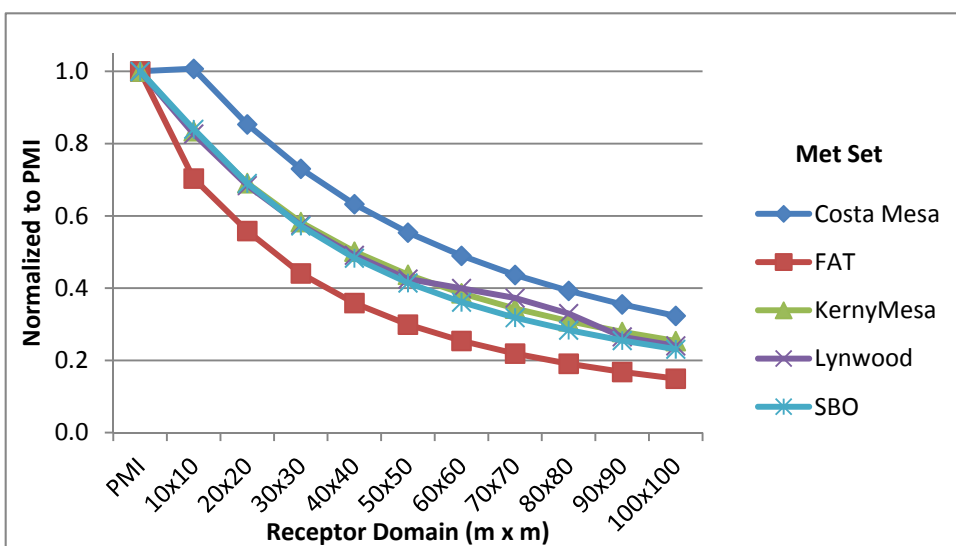


Figure 8.1

Large Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

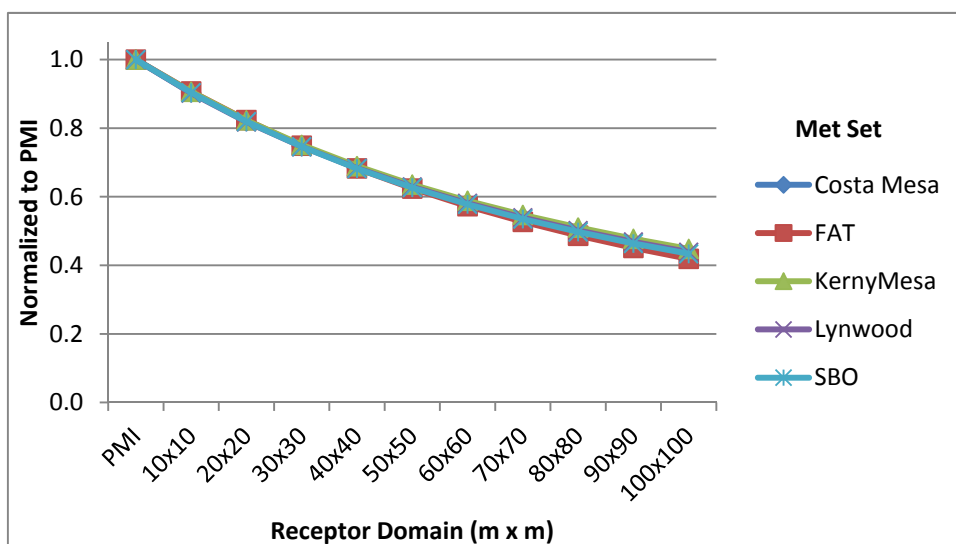


Figure 8.2

Medium Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

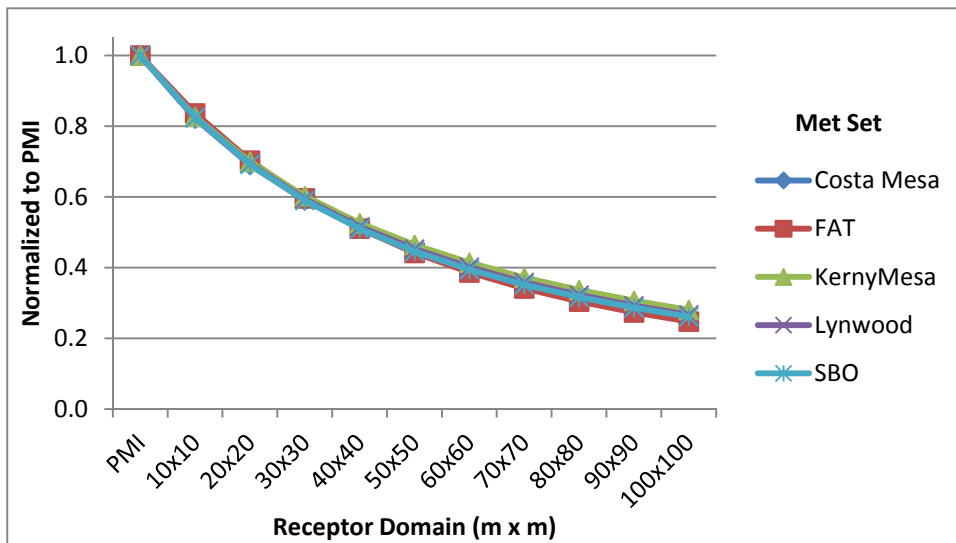


Figure 8.3

Small Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

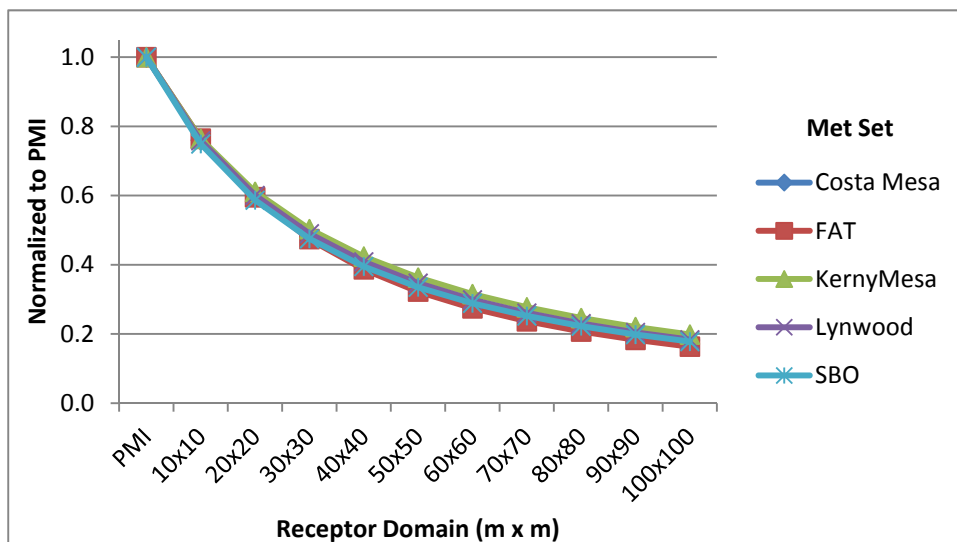


Figure 9.1

Large Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

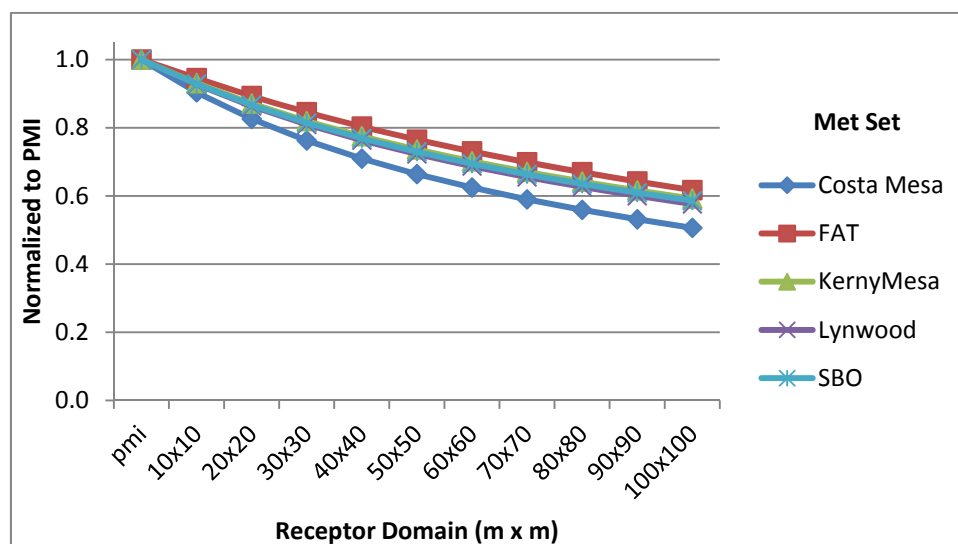


Figure 9.2

Medium Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

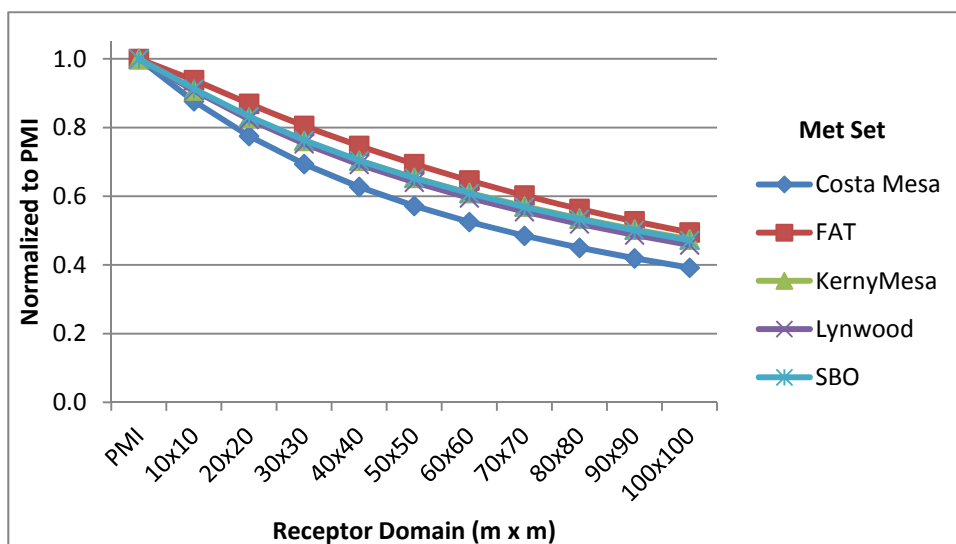


Figure 9.3

Small Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

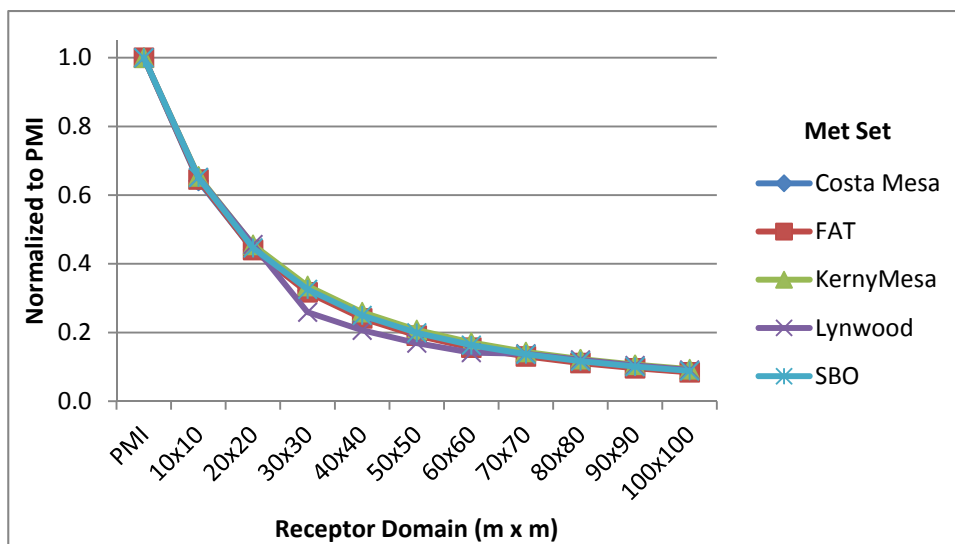


Figure 10.1

Large Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

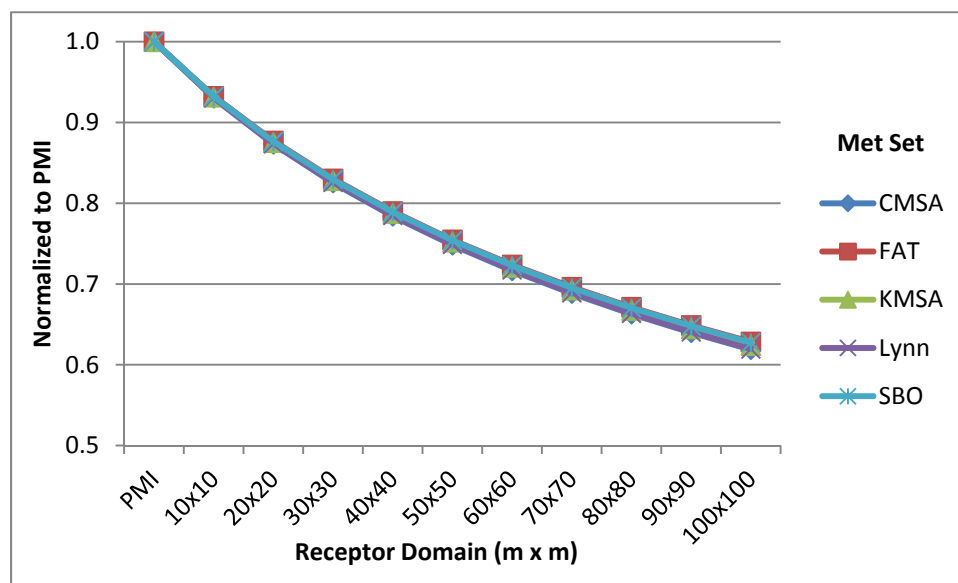
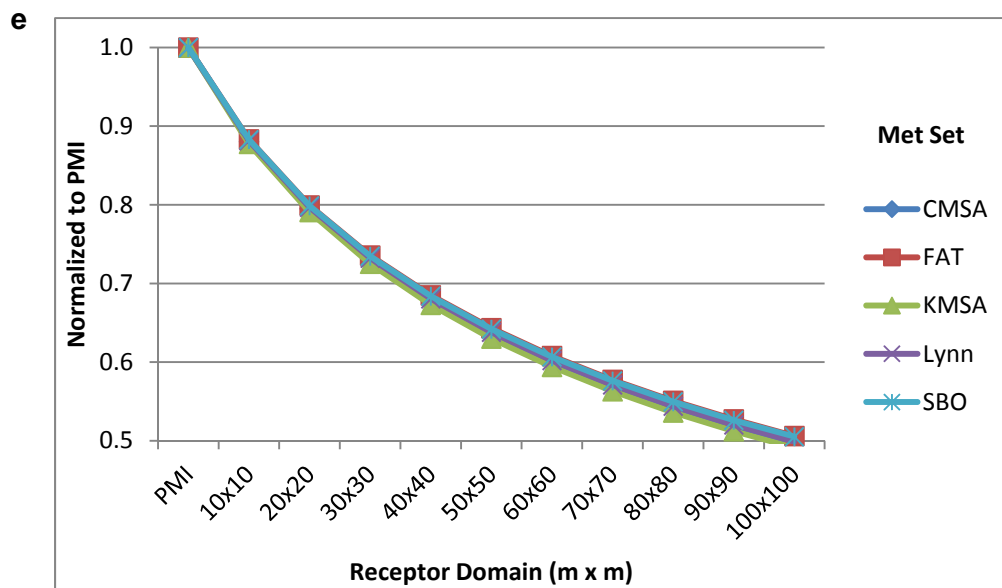


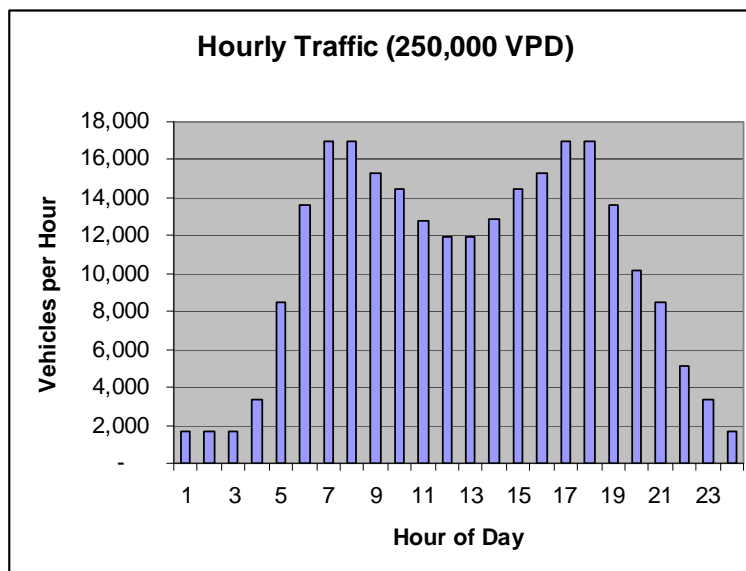
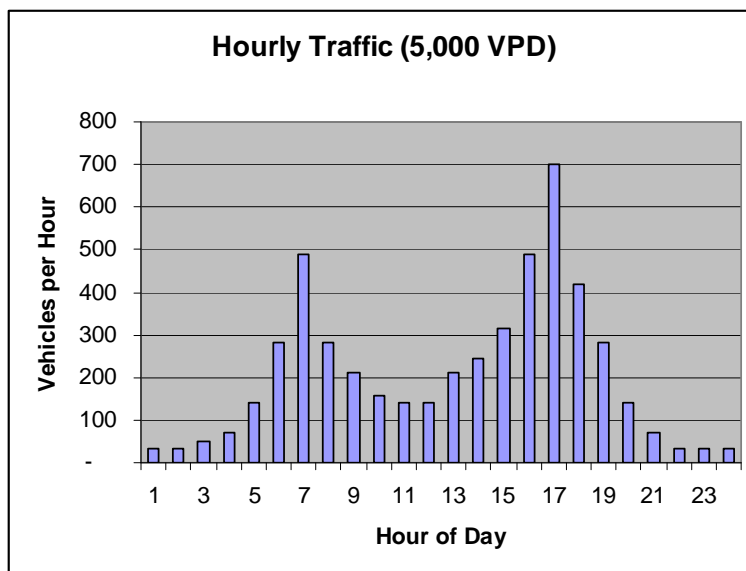
Figure 10.2

Small Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets



Appendix 1 – Hourly Variation for Traffic Line Source

Hour	5K VPD	250K VPD
1	35	1,700
2	35	1,700
3	49	1,700
4	70	3,400
5	140	8,500
6	280	13,600
7	490	17,000
8	280	17,000
9	210	15,300
10	156	14,450
11	140	12,750
12	140	11,900
13	210	11,900
14	245	12,850
15	315	14,450
16	490	15,300
17	700	17,000
18	420	17,000
19	280	13,600
20	140	10,200
21	70	8,500
22	35	5,100
23	35	3,400
24	35	1,700
Sum	5,000	250,000
Peak Hour	700	17,000



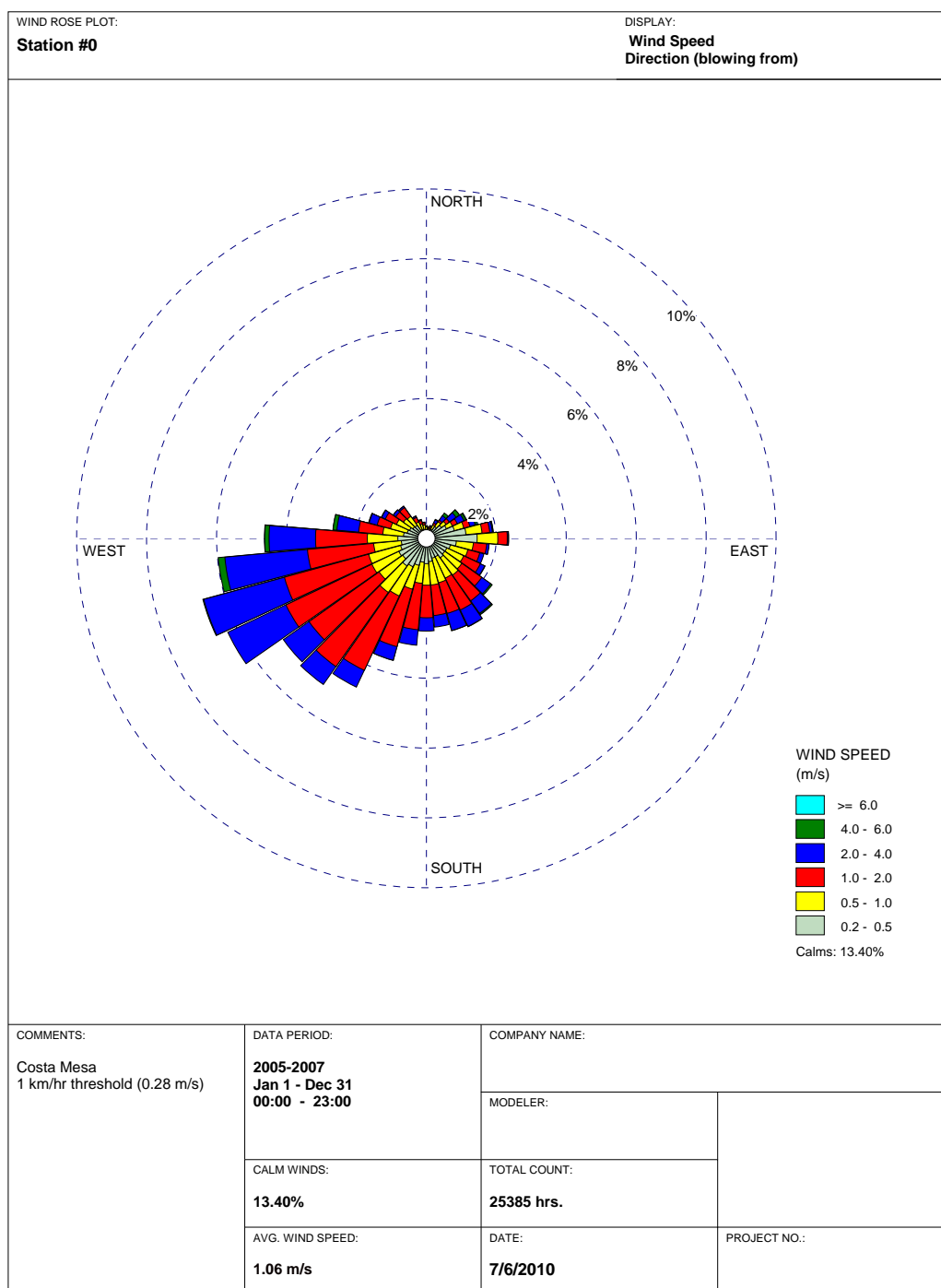
Appendix 2 – Meteorological Data

Figure Ap2.1
AERMET Data from Districts



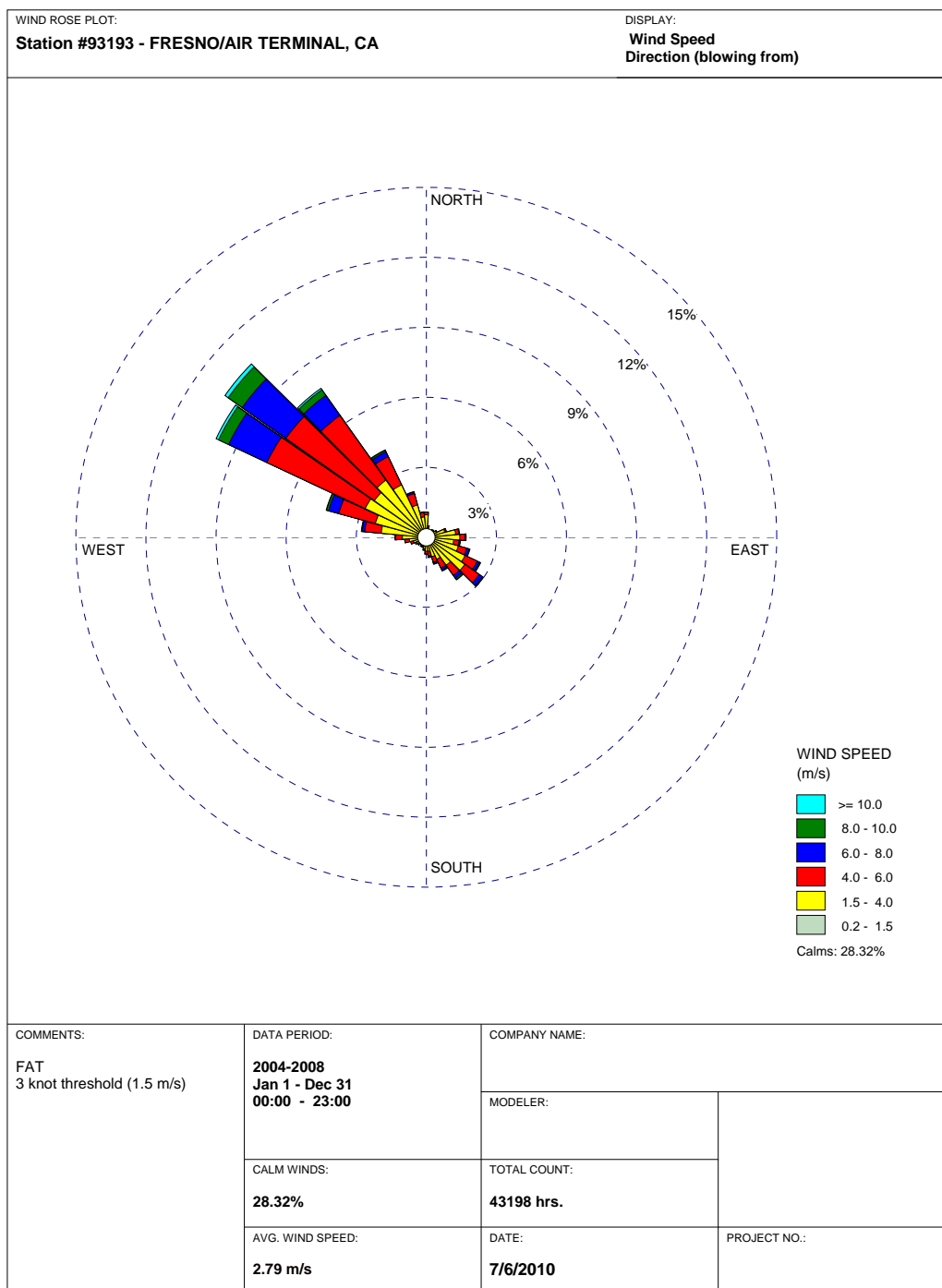
The above figure shows the locations where AERMET data are available from Districts. We selected the following stations for this analysis which include stations that are near the ocean and inland – Costa Mesa, Fresno Air Terminal (FAT), Kearny Mesa, Lynwood, and San Bernardino.

Figure AP2.2 – Costa Mesa – Wind Rose Summary



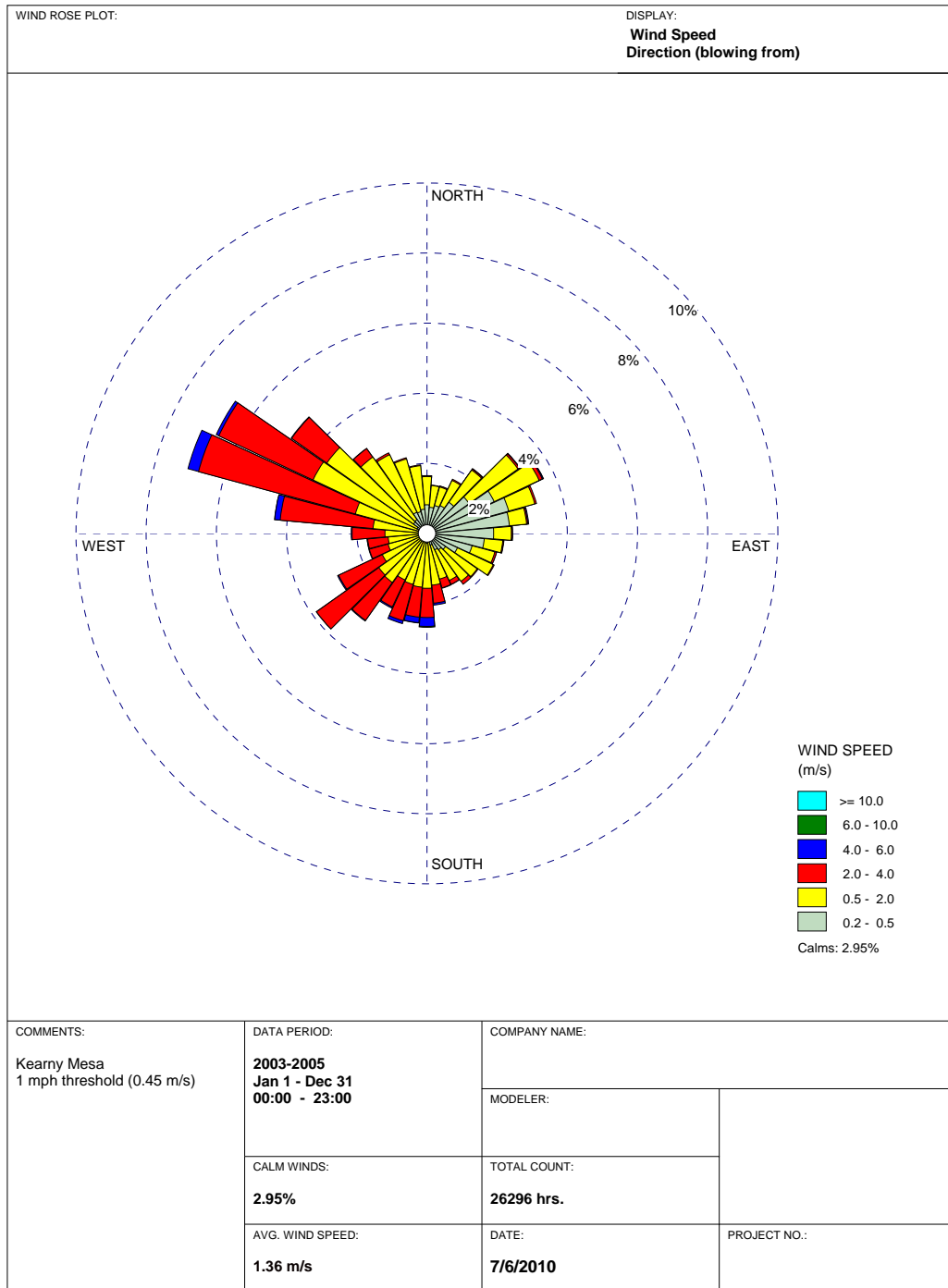
WRPLOT View - Lakes Environmental Software

Figure AP2.3 – Fresno Air Terminal – Wind Rose Summary



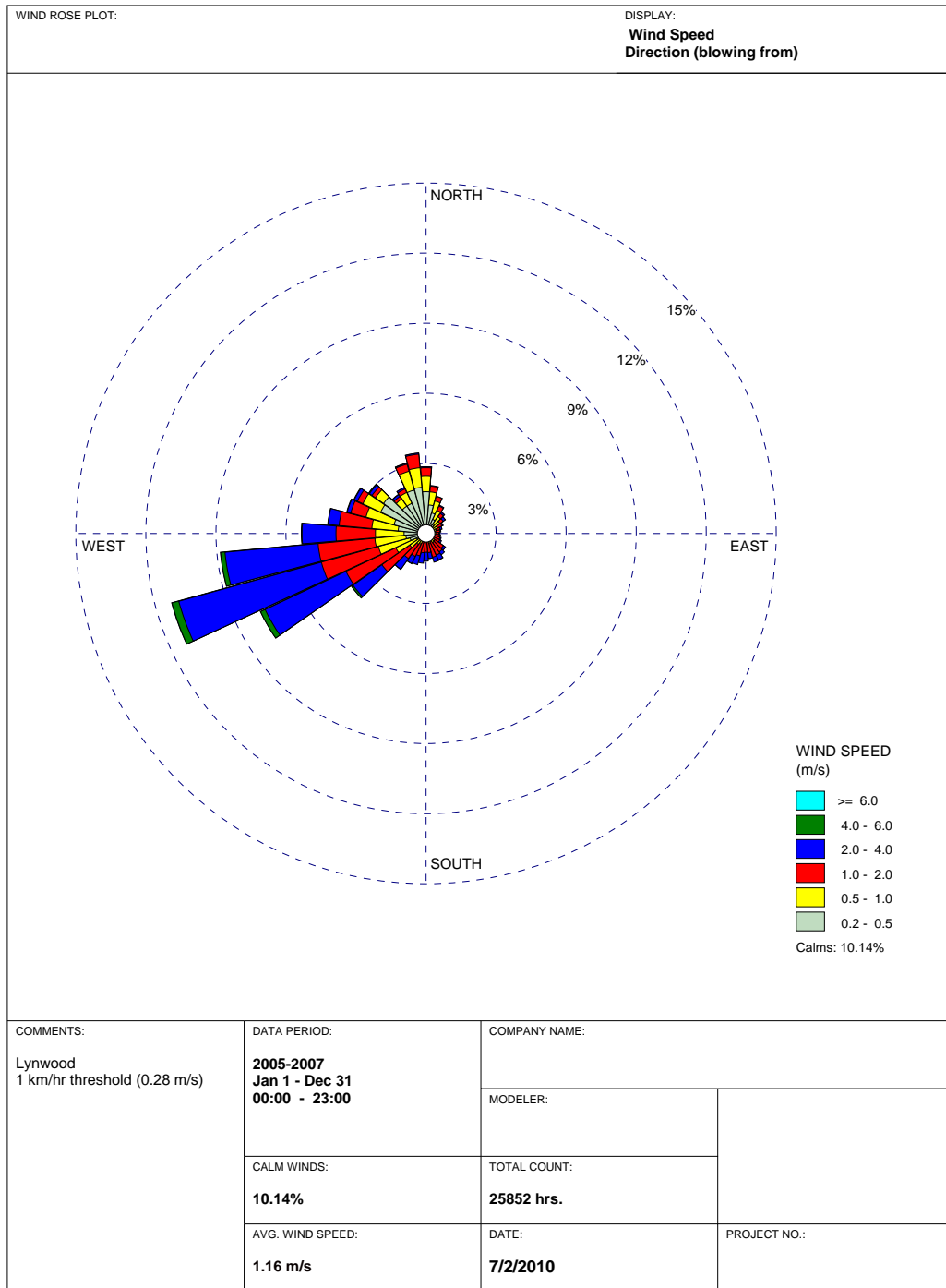
WRPLOT View - Lakes Environmental Software

Figure AP2.4 – Kearny Mesa – Wind Rose Summary



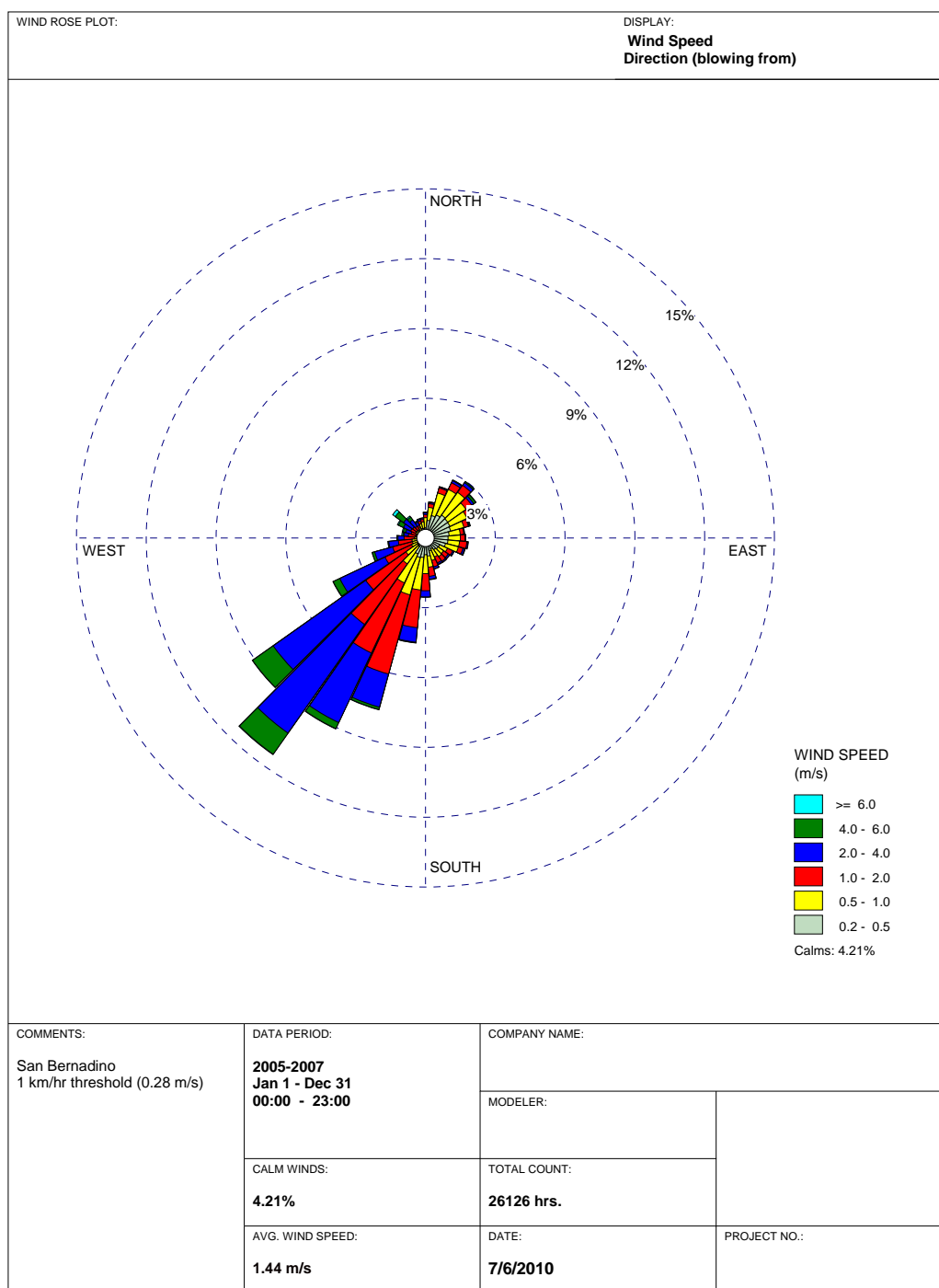
WRPLOT View - Lakes Environmental Software

Figure AP2.5 – Lynwood – Wind Rose Summary



WRPLOT View - Lakes Environmental Software

Figure AP2.6 – San Bernardino – Wind Rose Summary



WRPLOT View - Lakes Environmental Software

Appendix 3 – Sources, Receptors, Concentrations

Figure AP3.1.1 – Large Point Source – Costa Mesa

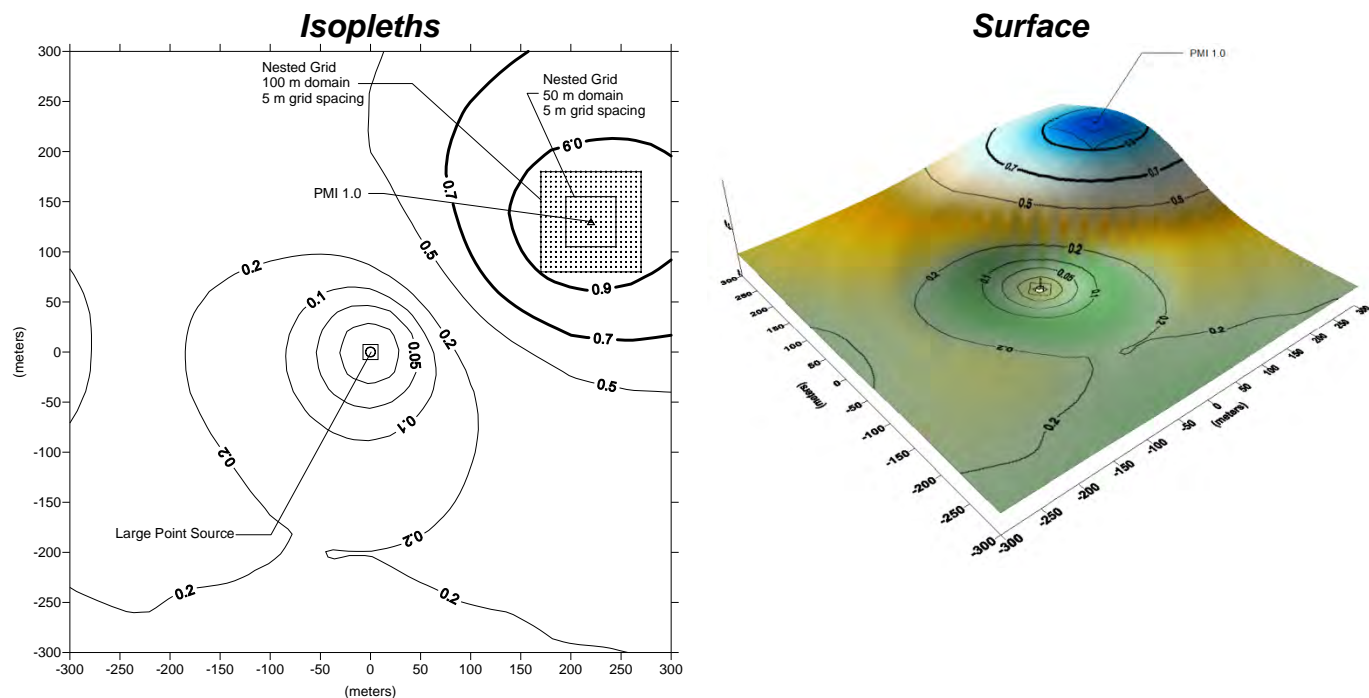


Figure AP3.1.2 – Large Point Source – Fresno Air Terminal

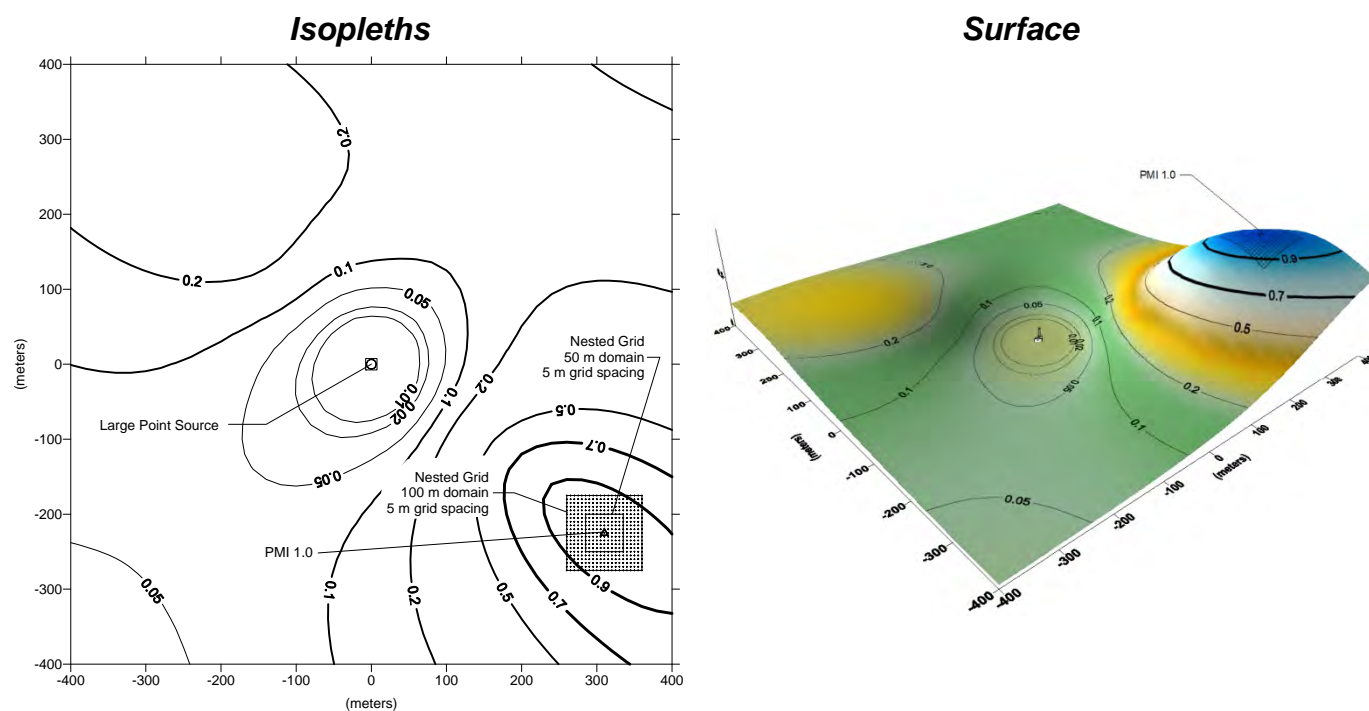


Figure AP3.1.3 – Large Point Source – Kearny Mesa

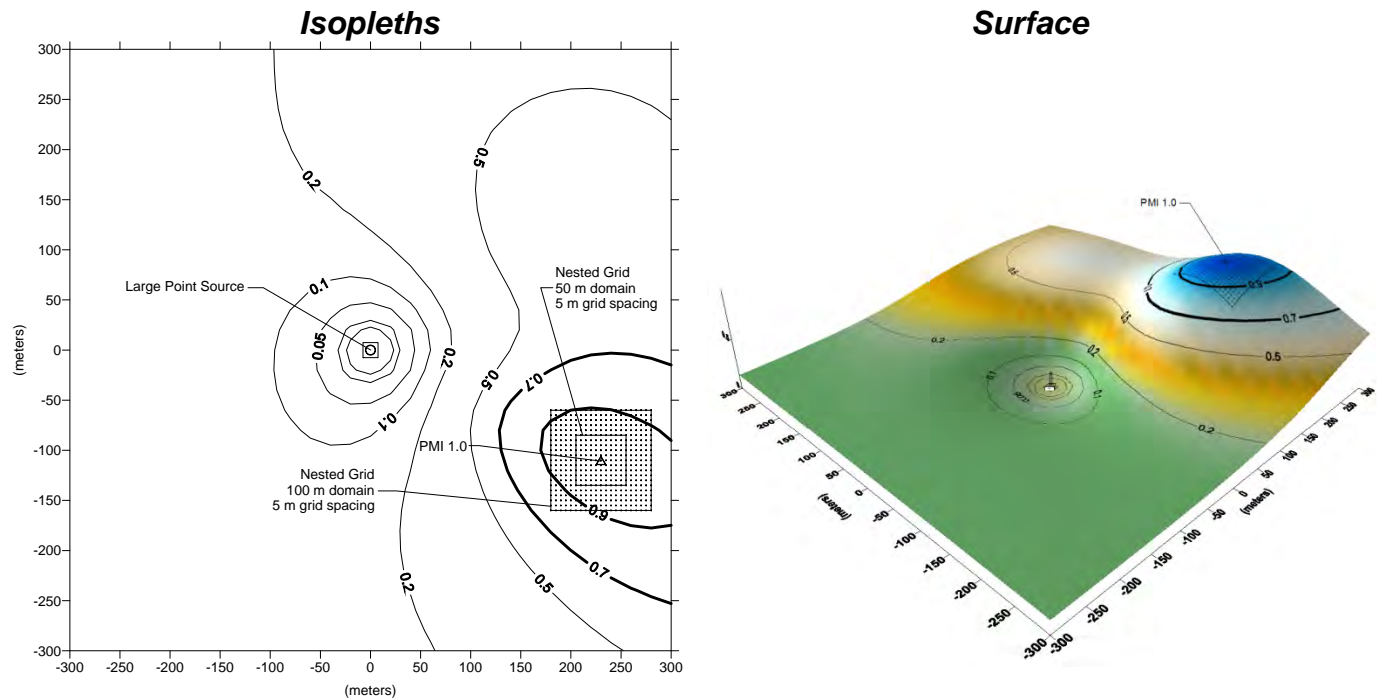


Figure AP3.1.4 – Large Point Source – Lynwood

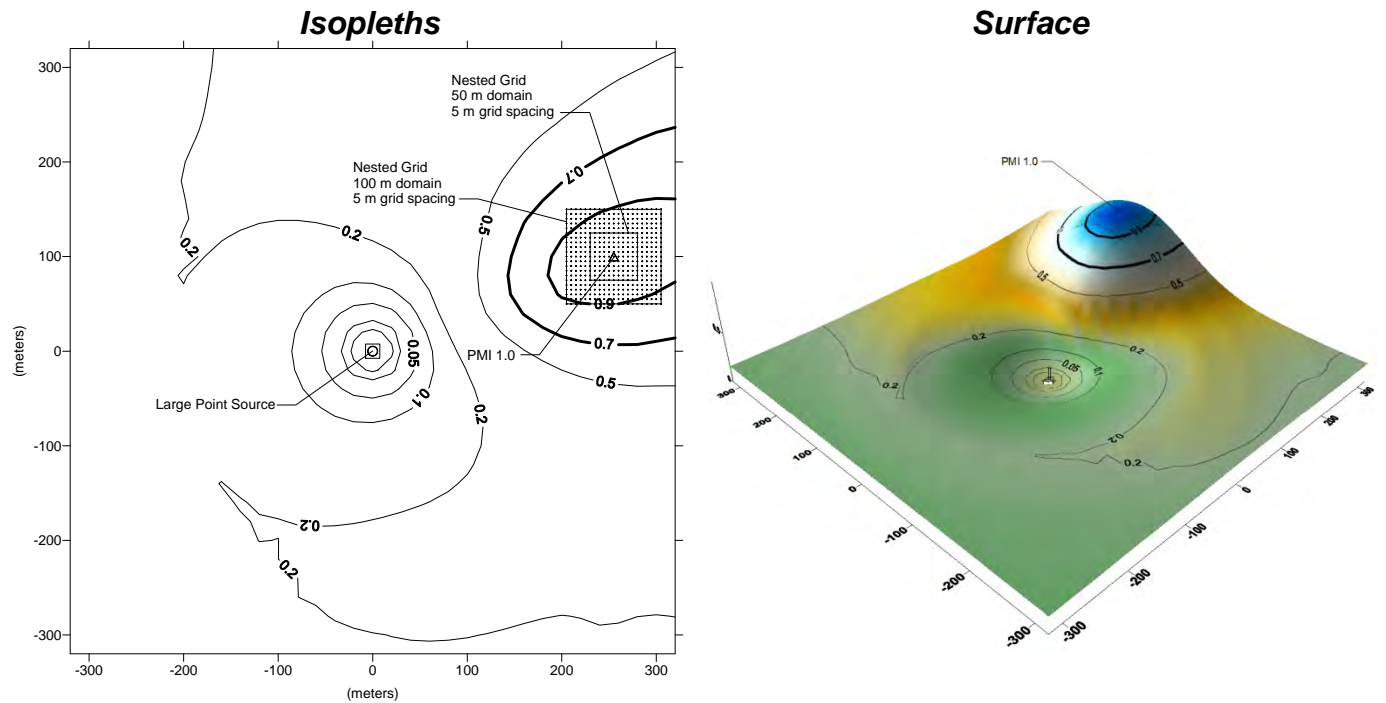


Figure AP3.1.5 – Large Point Source – San Bernardino

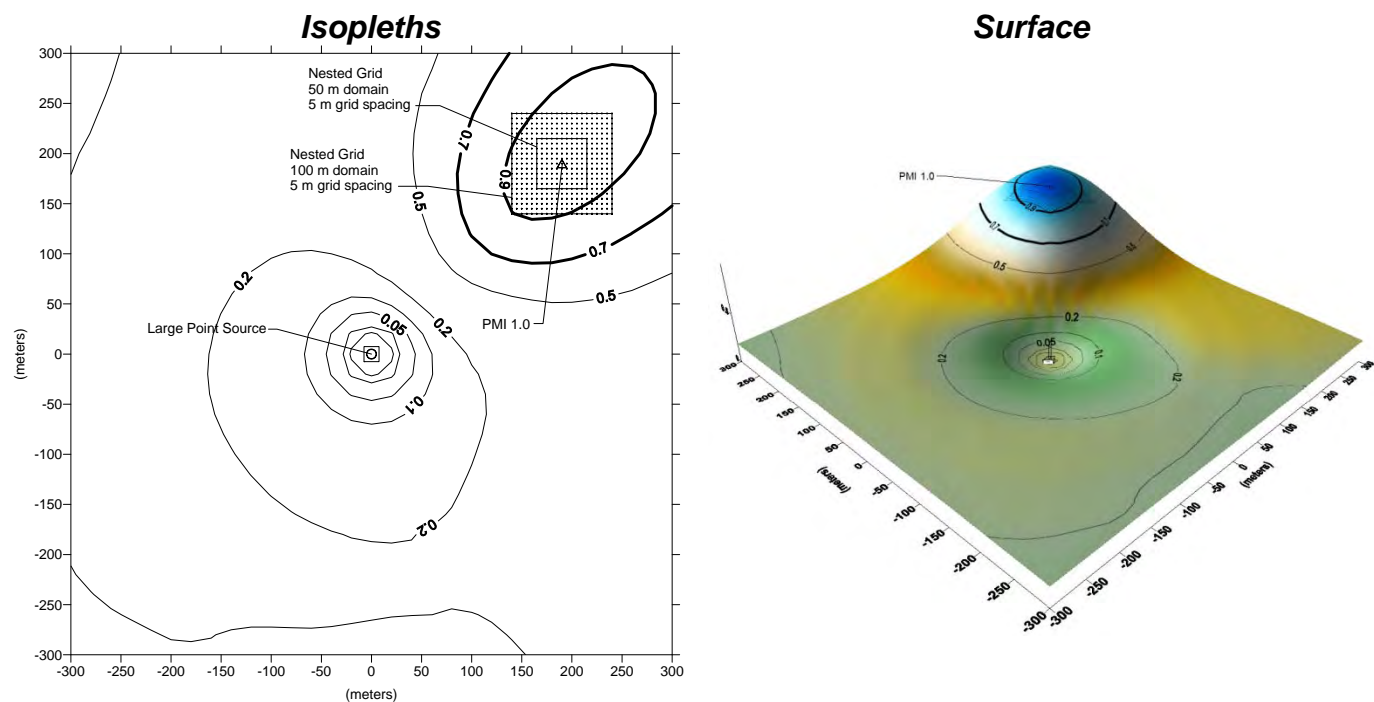


Figure AP3.2.1 – Medium Point Source – Costa Mesa

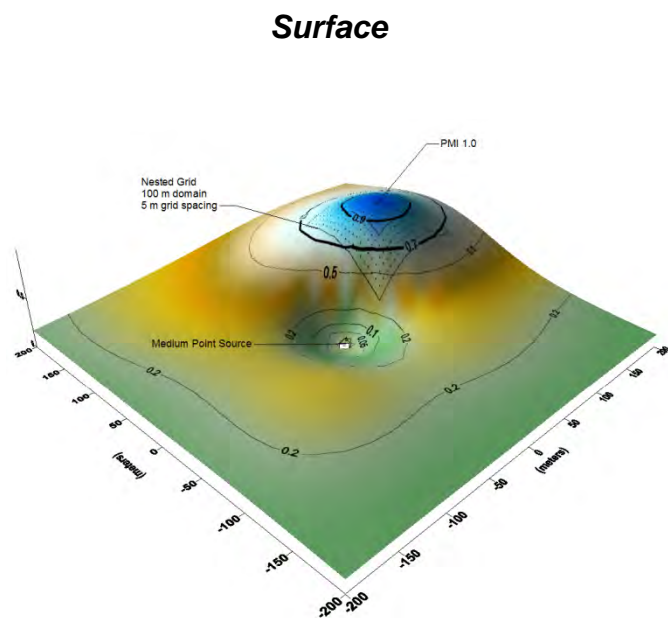
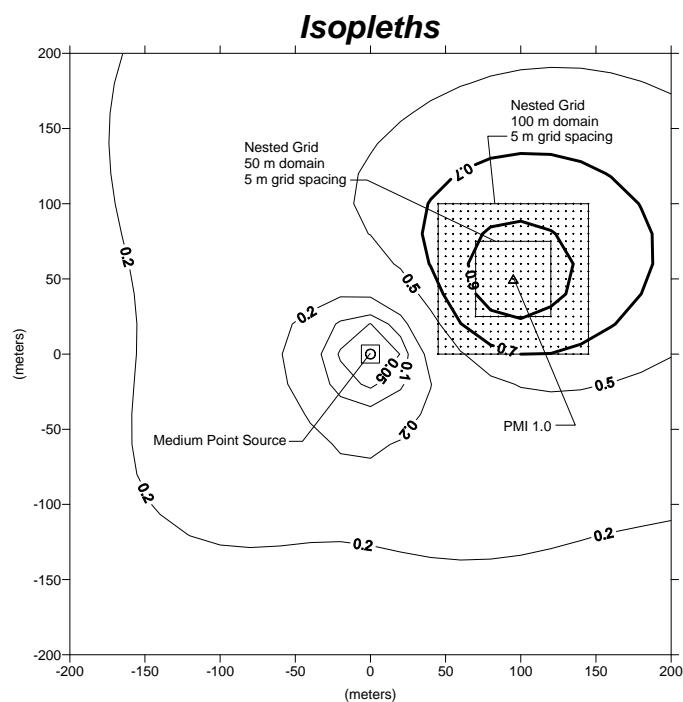


Figure AP3.2.2 – Medium Point Source – Fresno Air Terminal

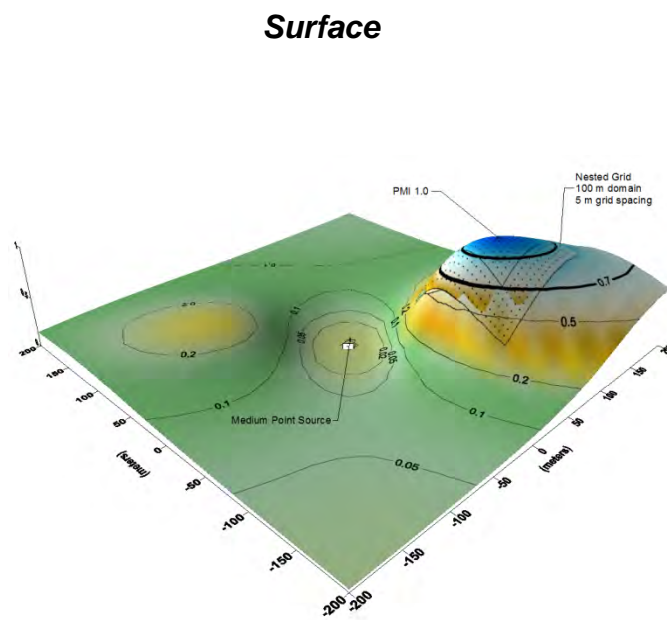
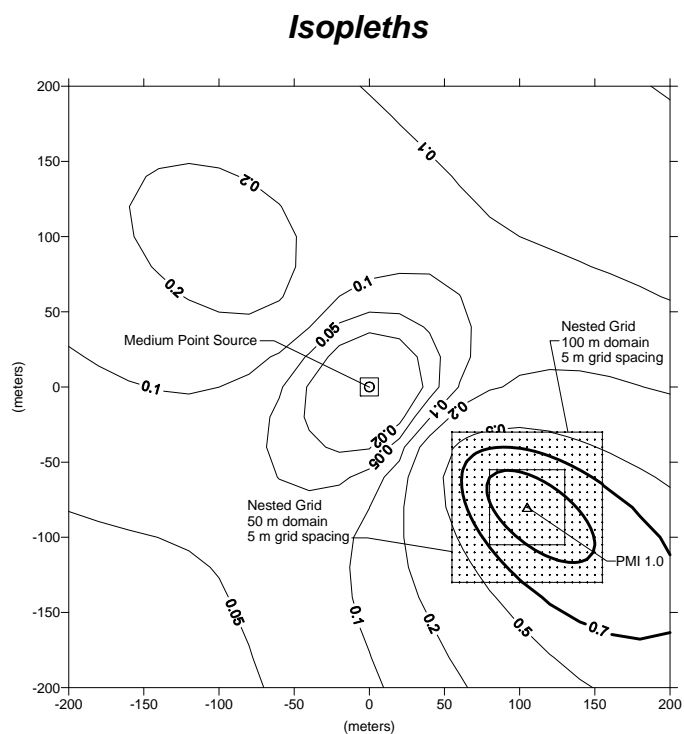


Figure AP3.2.3 – Medium Point Source – Kearny Mesa

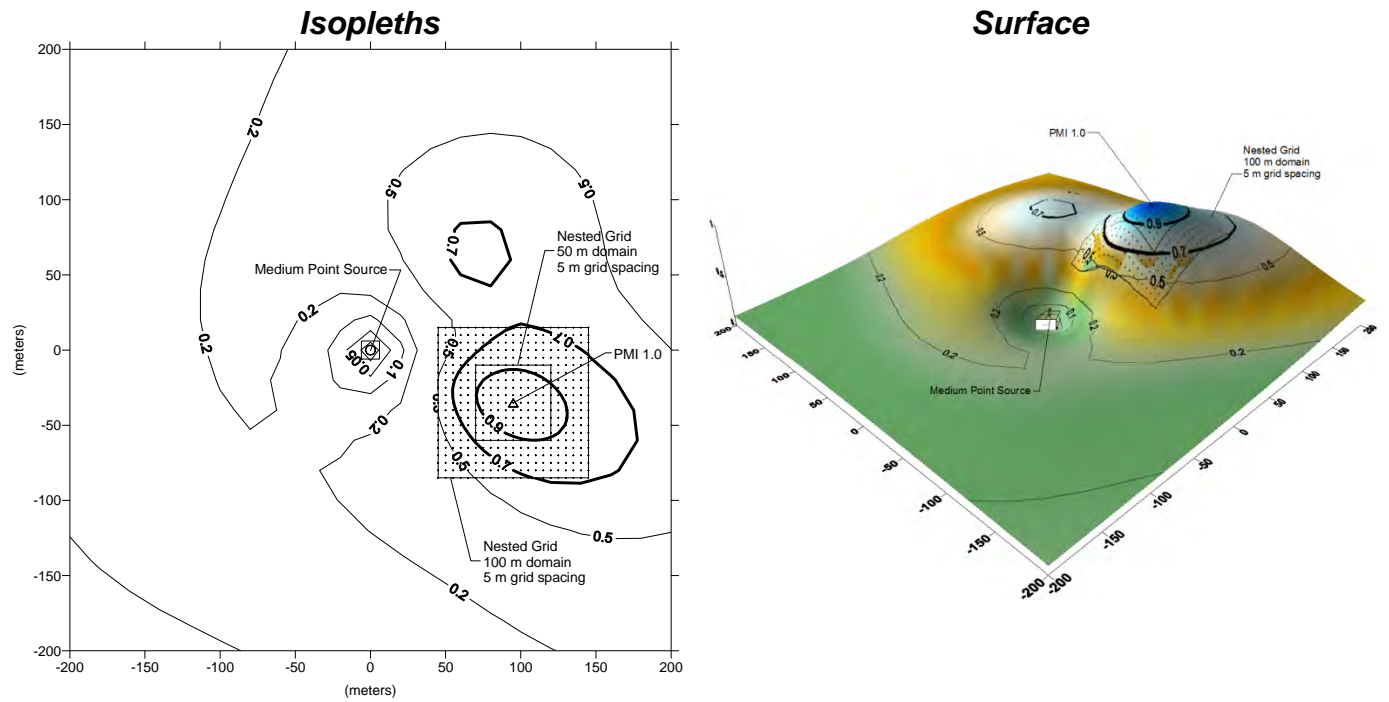


Figure AP3.2.4 – Medium Point Source – Lynwood

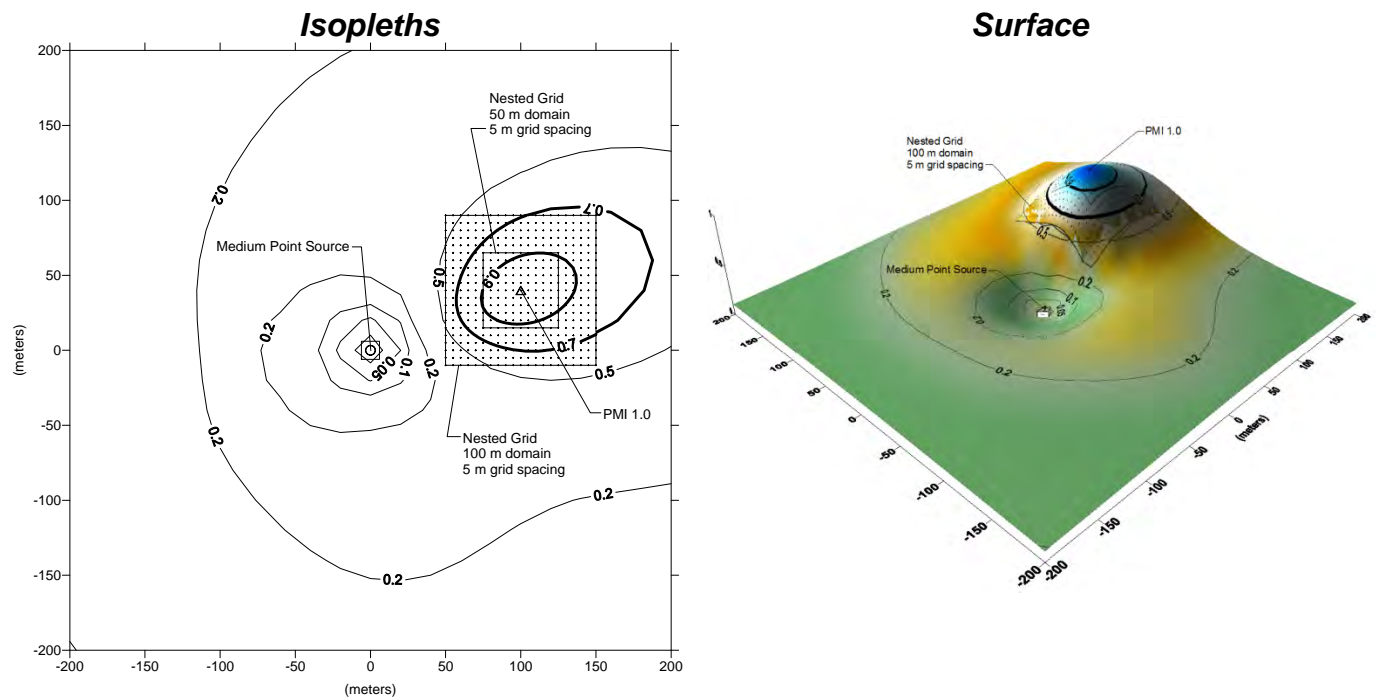


Figure AP3.2.5 – Medium Point Source – San Bernardino

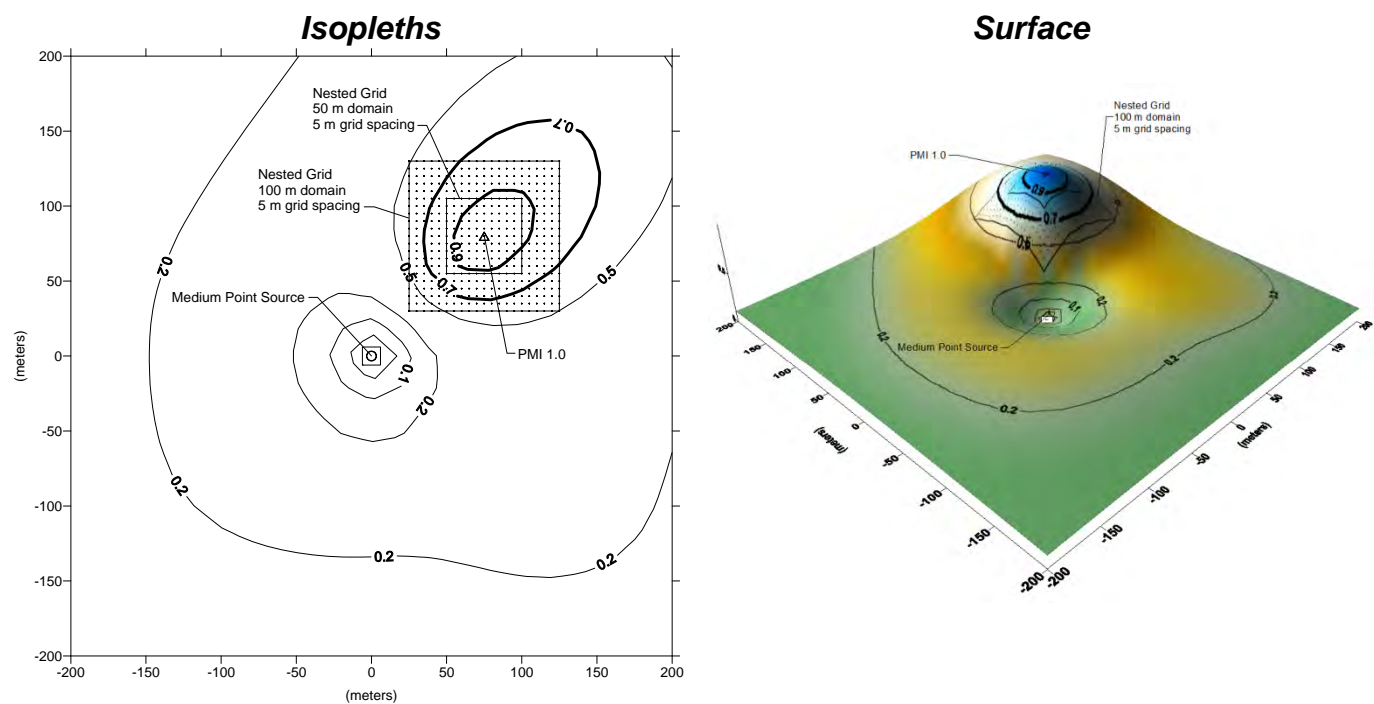


Figure AP3.3.1 – Small Point Source – Costa Mesa

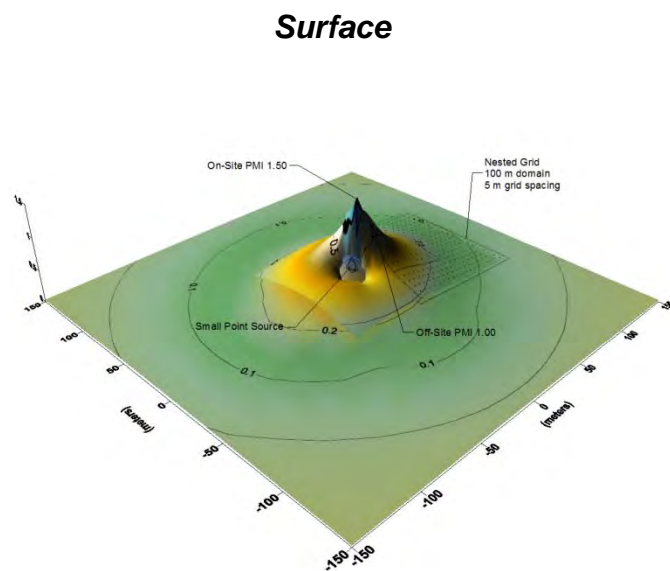
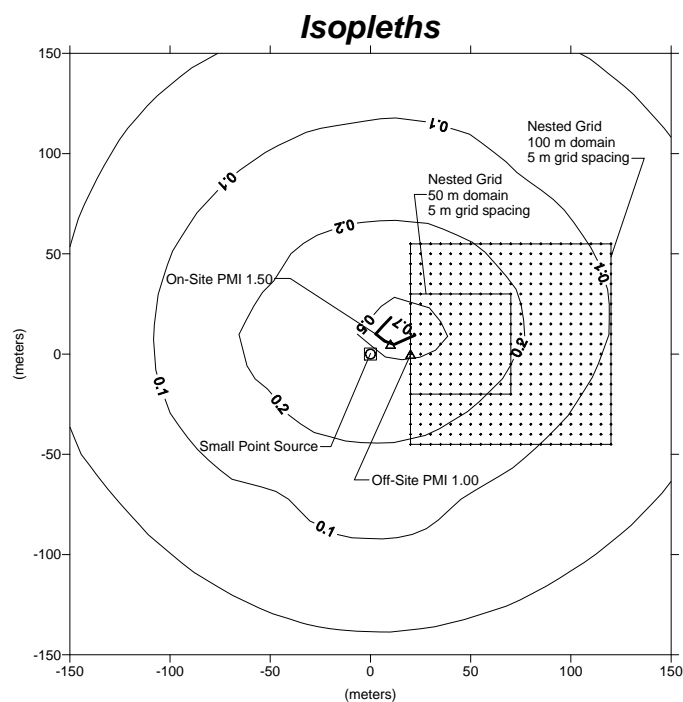


Figure AP3.3.2 – Small Point Source – Fresno Air Terminal

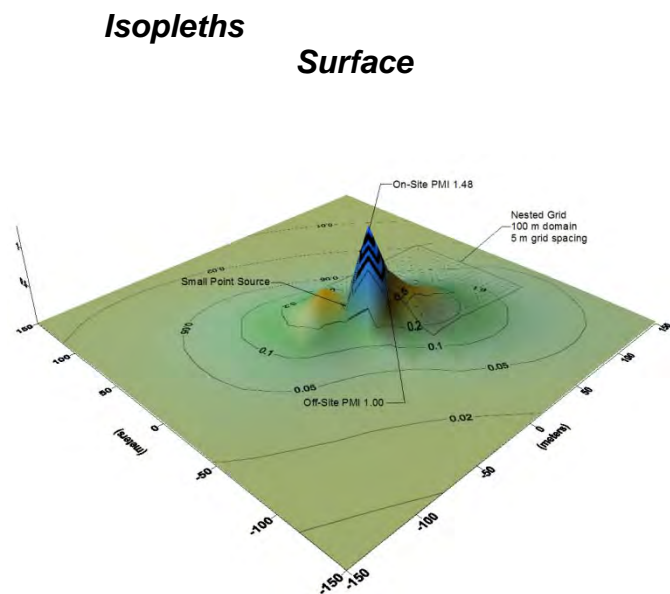
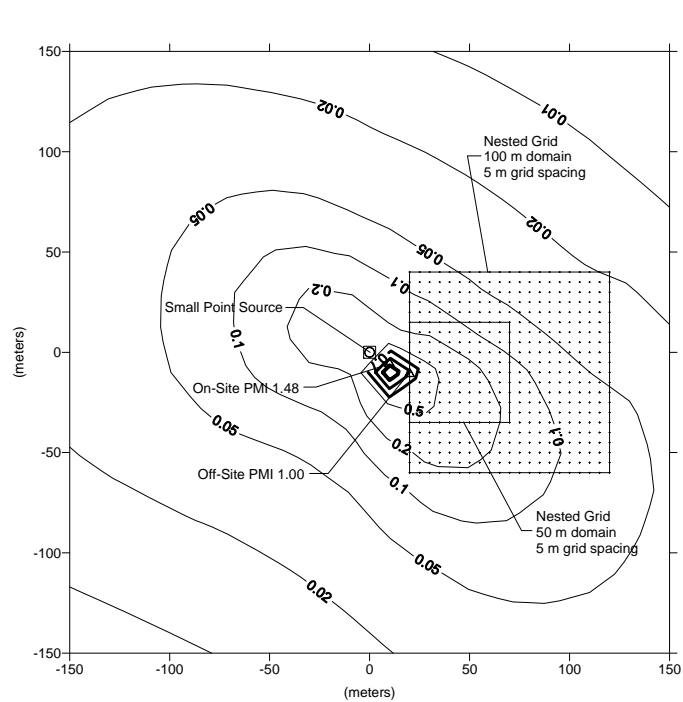


Figure AP3.3.3 – Small Point Source – Kearny Mesa

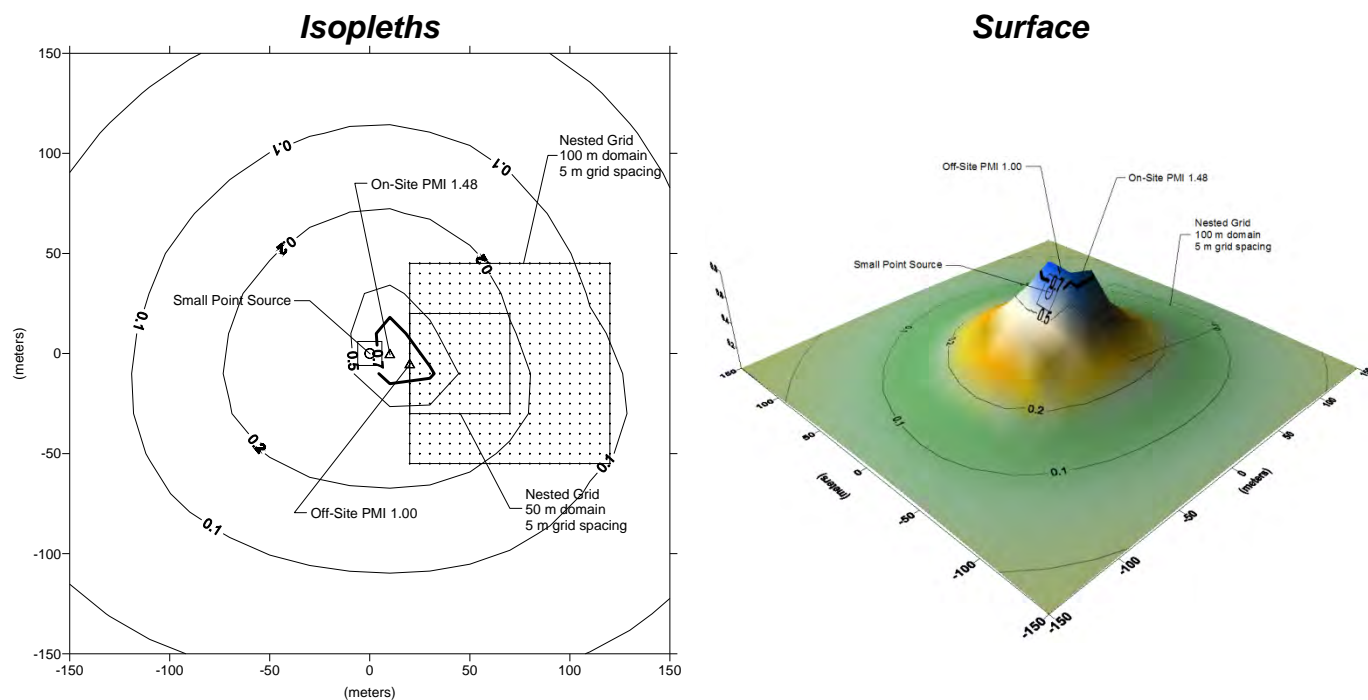


Figure AP3.3.4 – Small Point Source – Lynwood

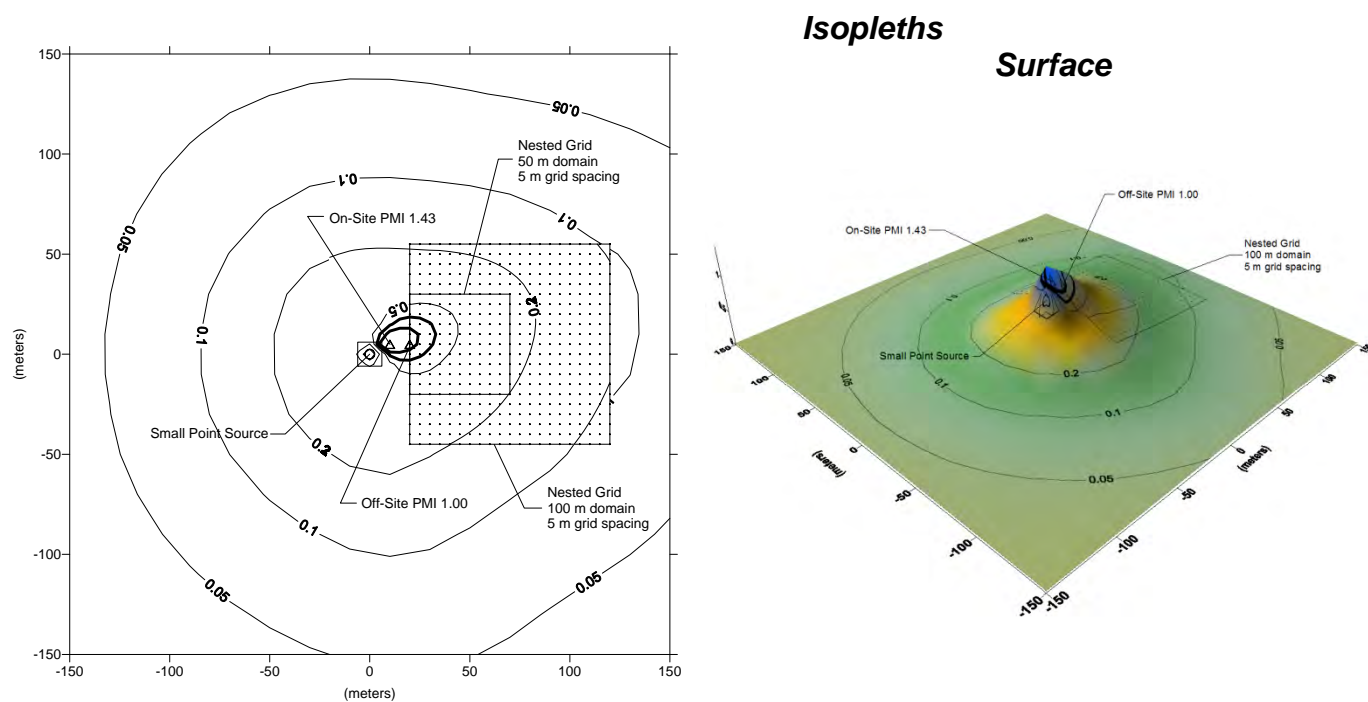


Figure AP3.3.5 – Small Point Source – San Bernardino

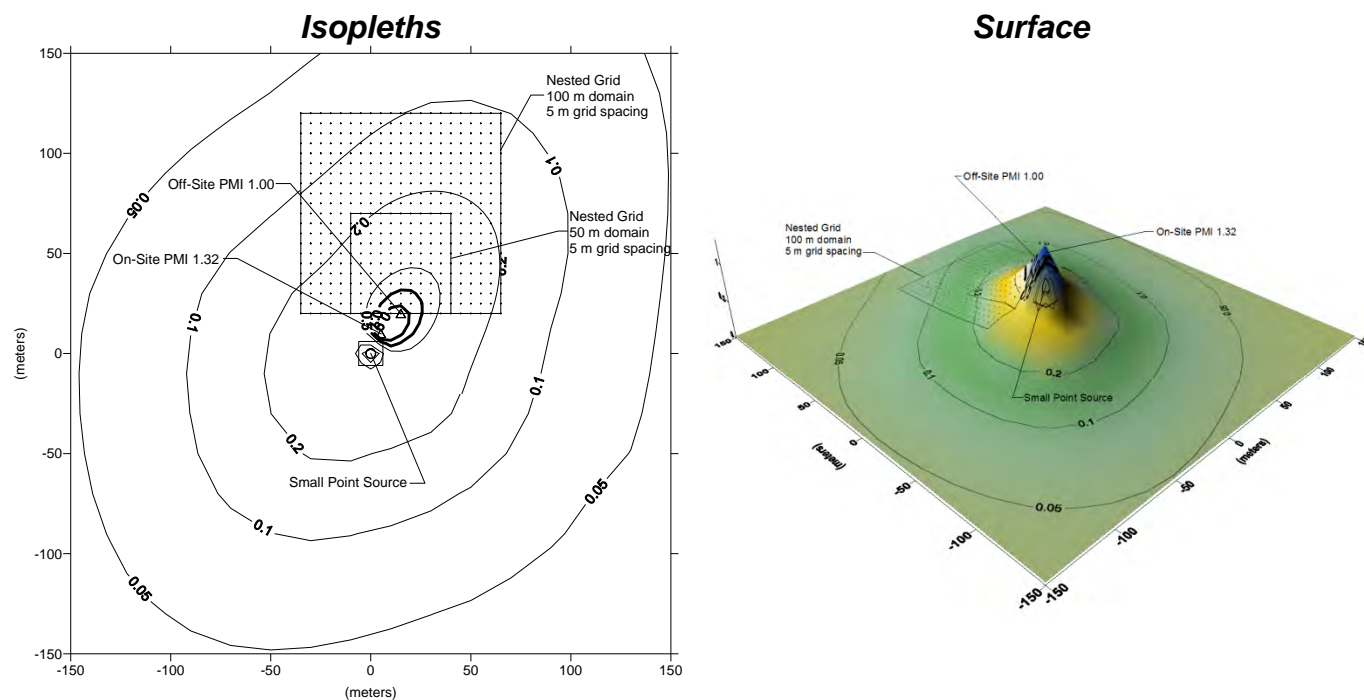


Figure AP3.4.1 – Large Volume Source – Costa Mesa

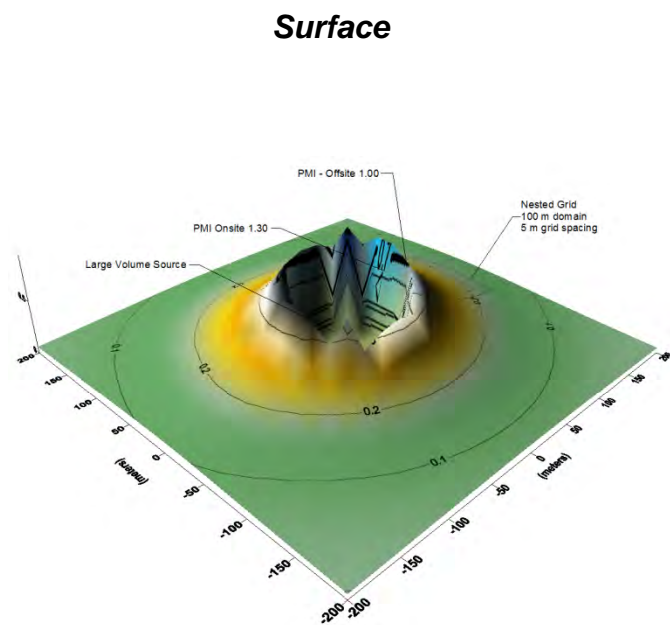
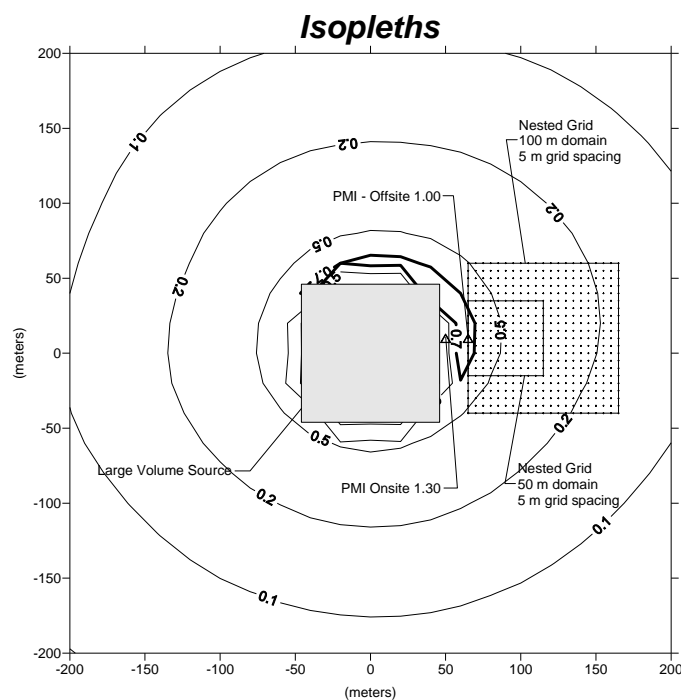


Figure AP3.4.2 – Large Volume Source – Fresno Air Terminal

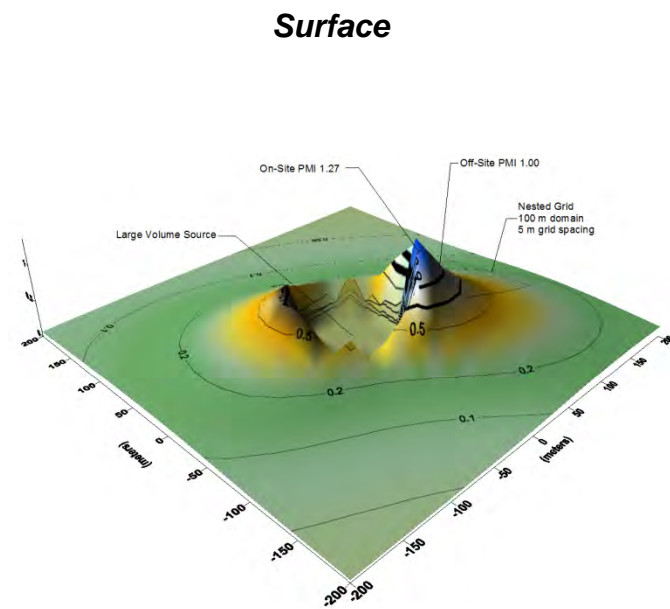
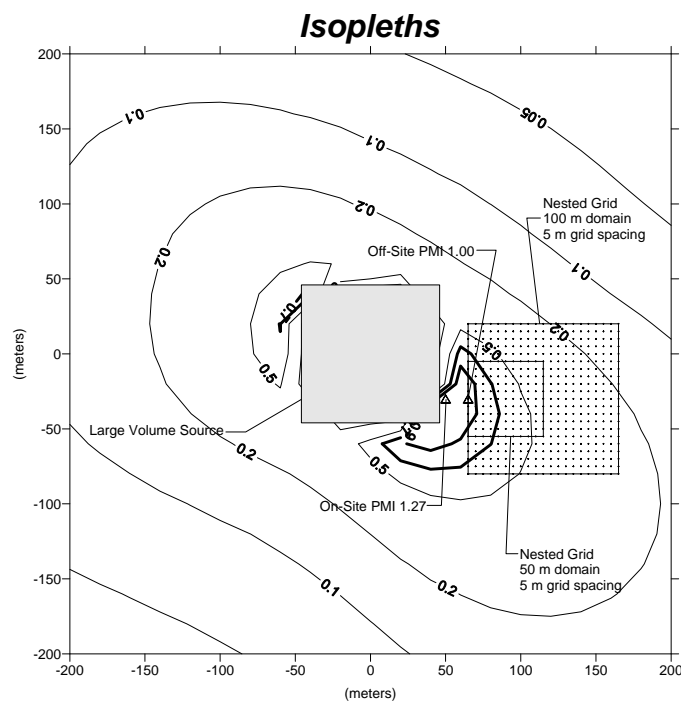


Figure AP3.4.3 – Large Volume Source – Kearny Mesa

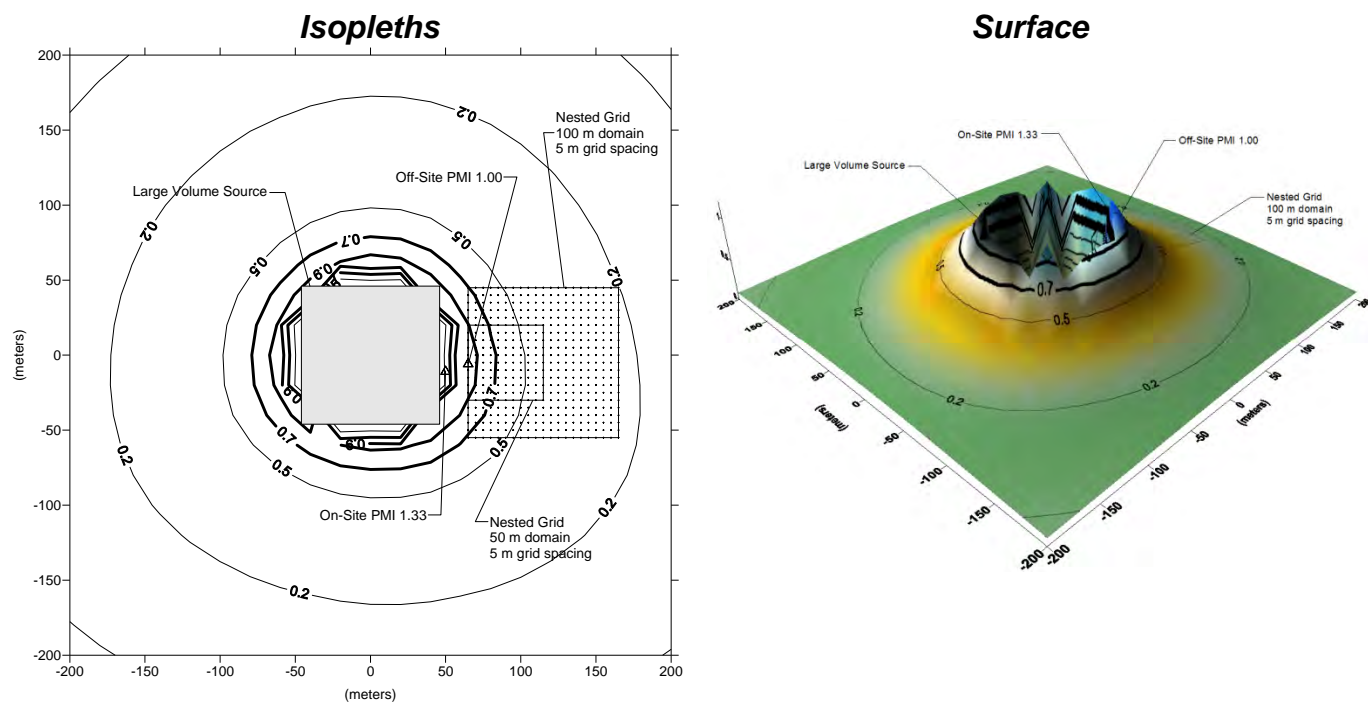


Figure AP3.4.4 – Large Volume Source – Lynwood

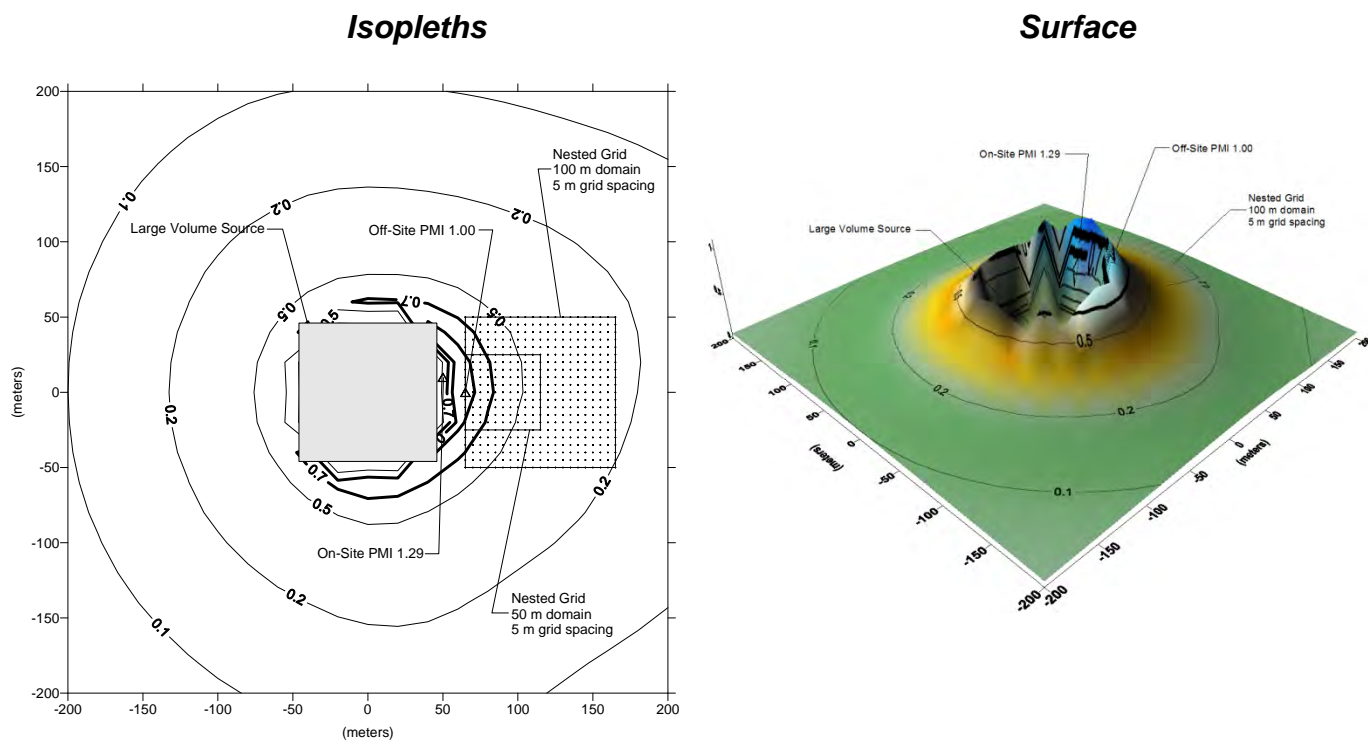


Figure AP3.4.5 – Large Volume Source – San Bernardino

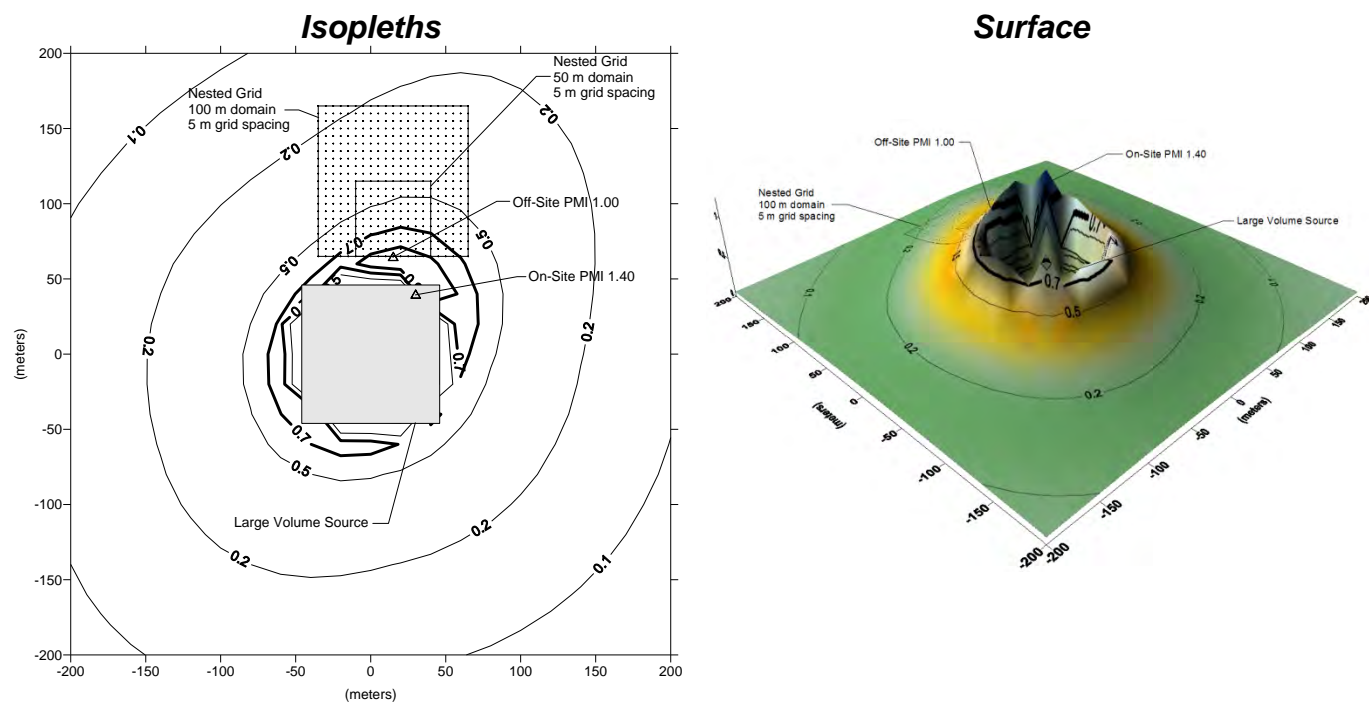


Figure AP3.5.1 – Medium Volume Source – Costa Mesa

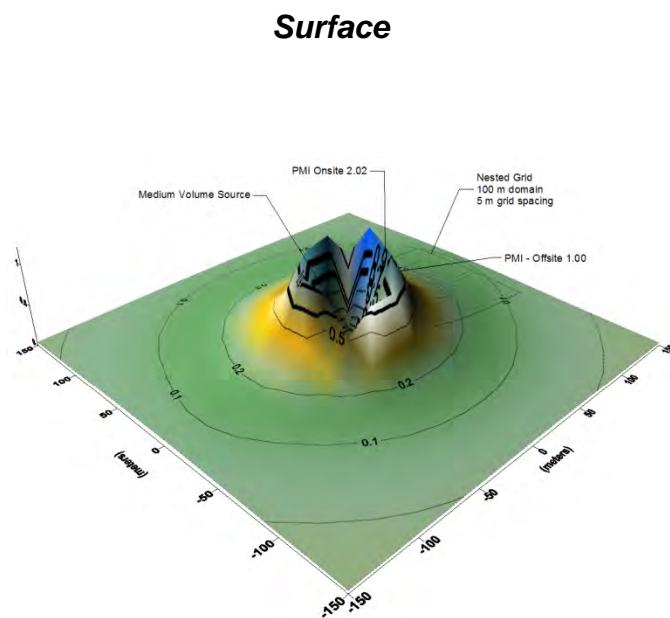
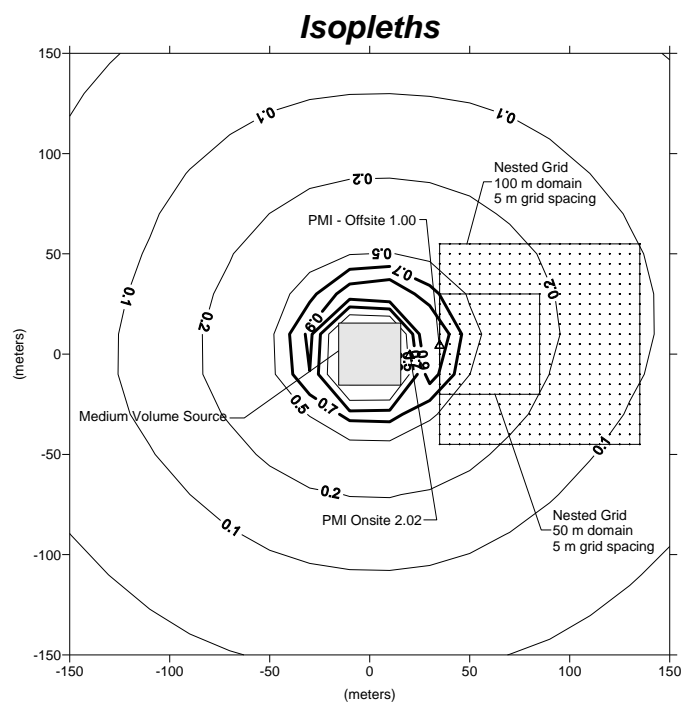


Figure AP3.5.2 – Medium Volume Source – Fresno Air Terminal

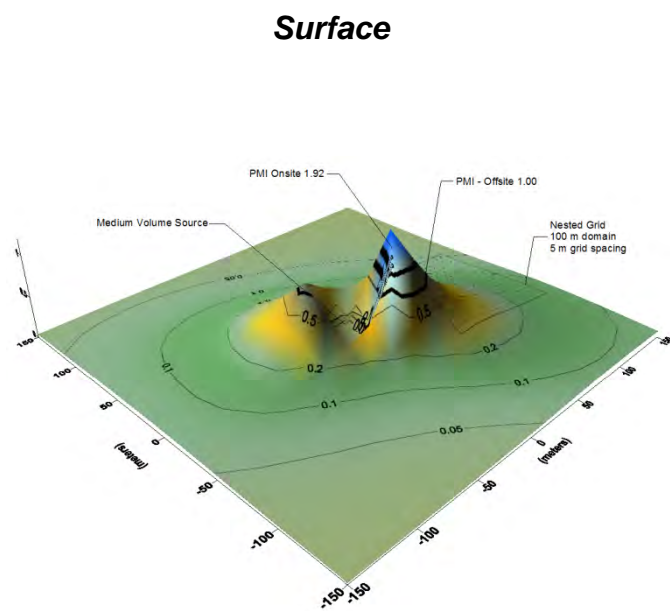
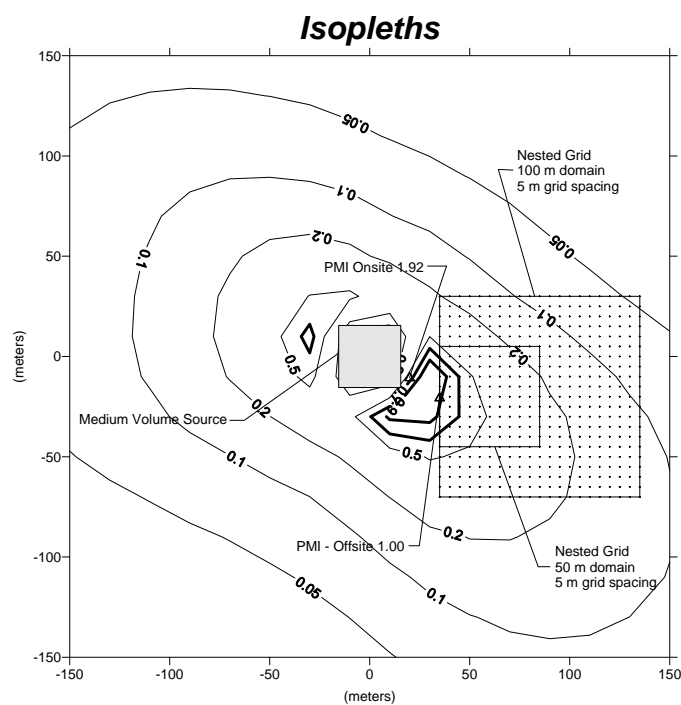


Figure AP3.5.3 – Medium Volume Source – Kearny Mesa

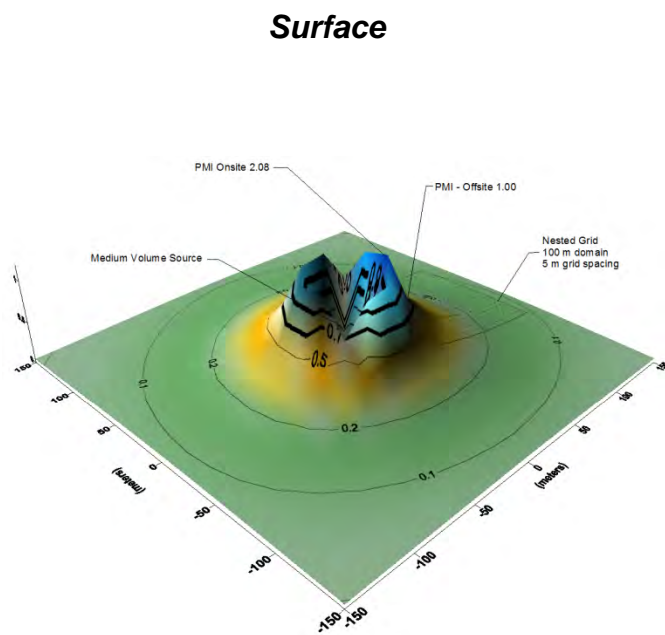
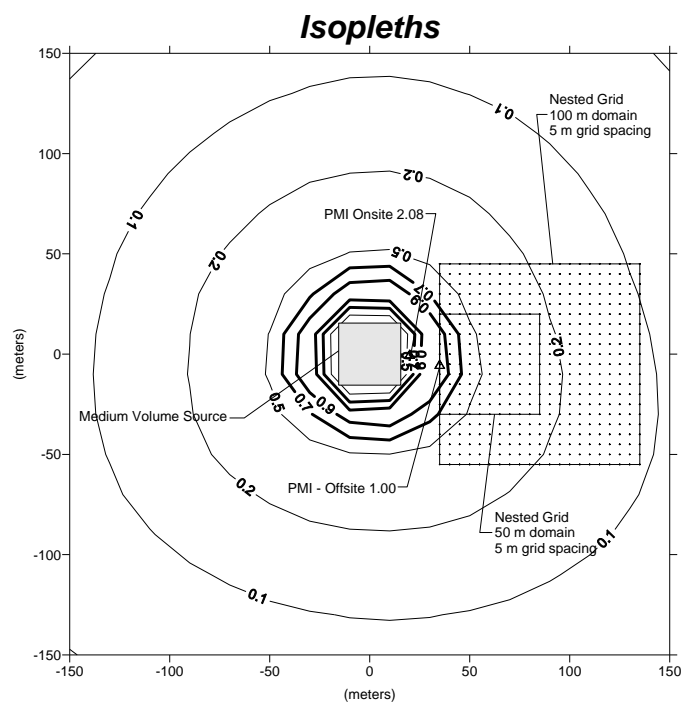


Figure AP3.5.4 – Medium Volume Source – Lynnwood

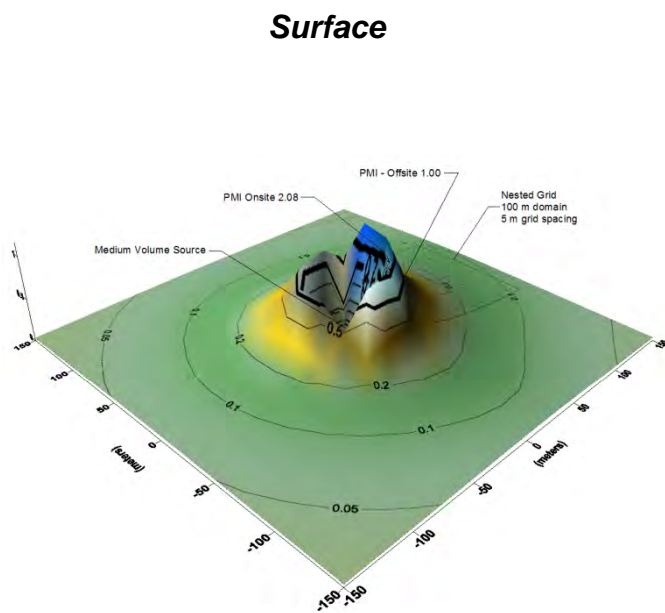
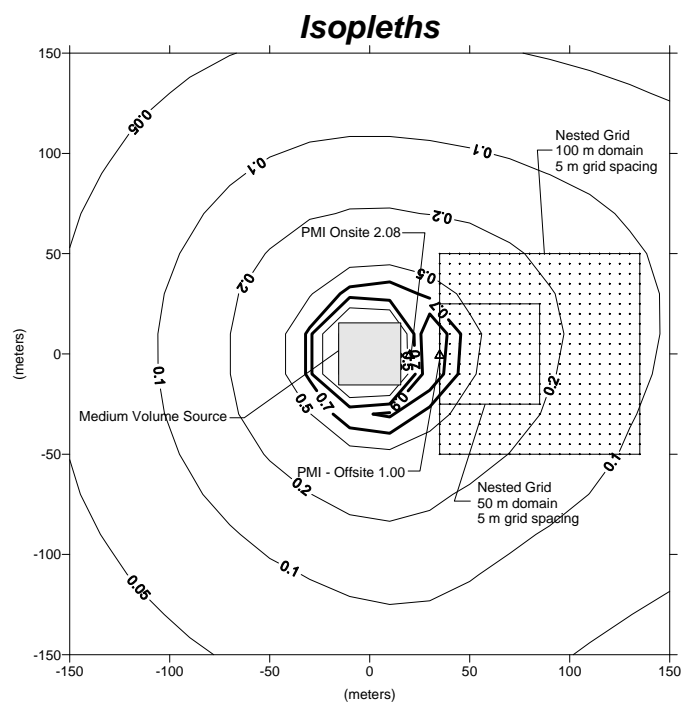


Figure AP3.5.5 – Medium Volume Source – San Bernardino

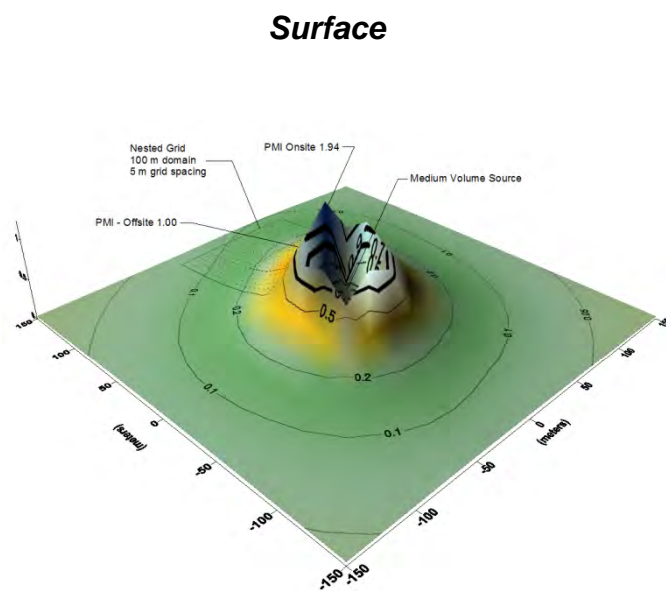
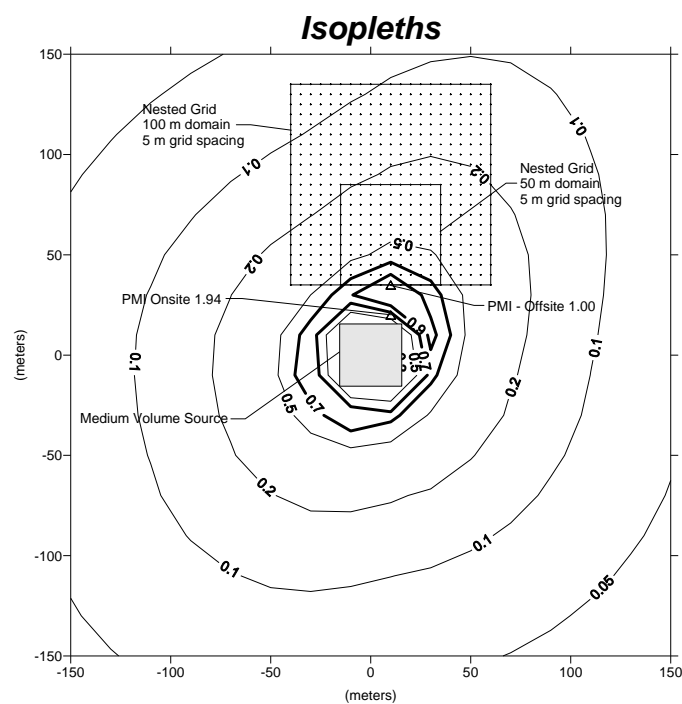


Figure AP3.6.1 – Small Volume Source – Costa Mesa

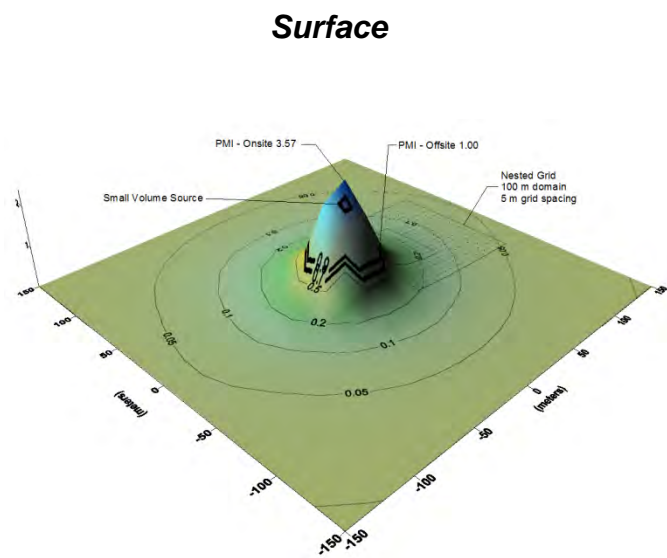
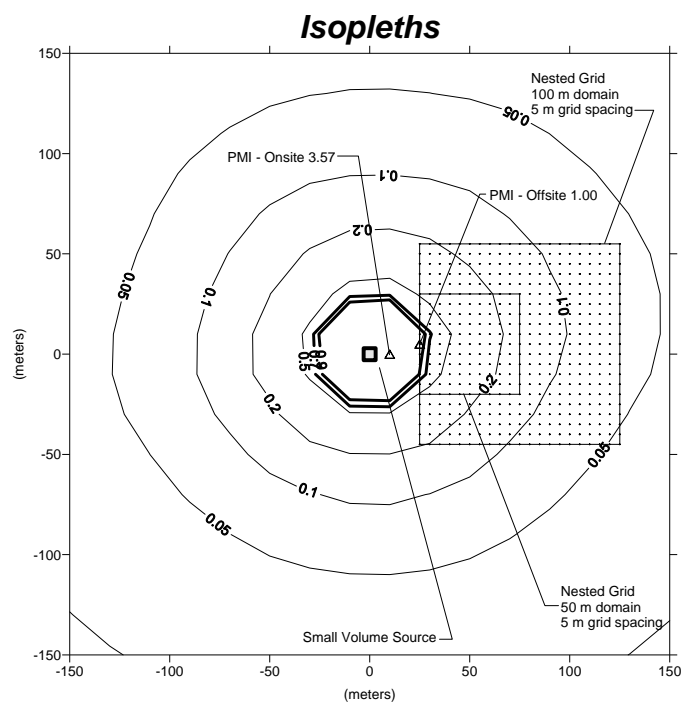


Figure AP3.6.2 – Small Volume Source – Fresno Air Terminal

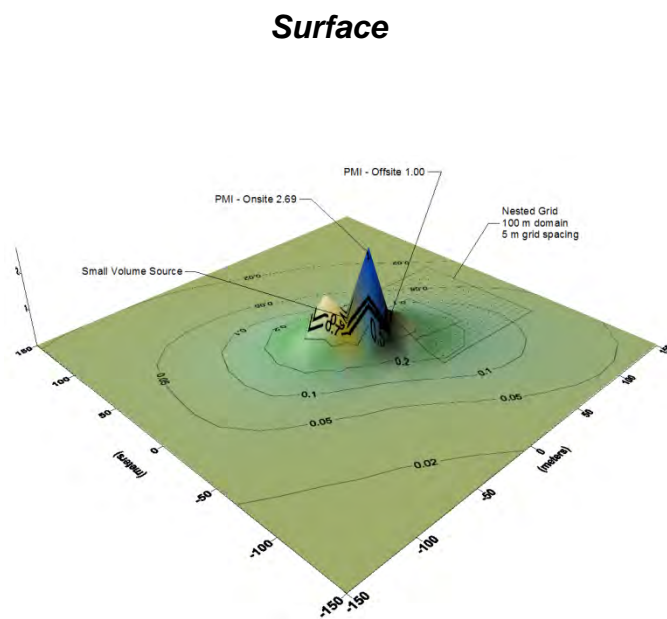
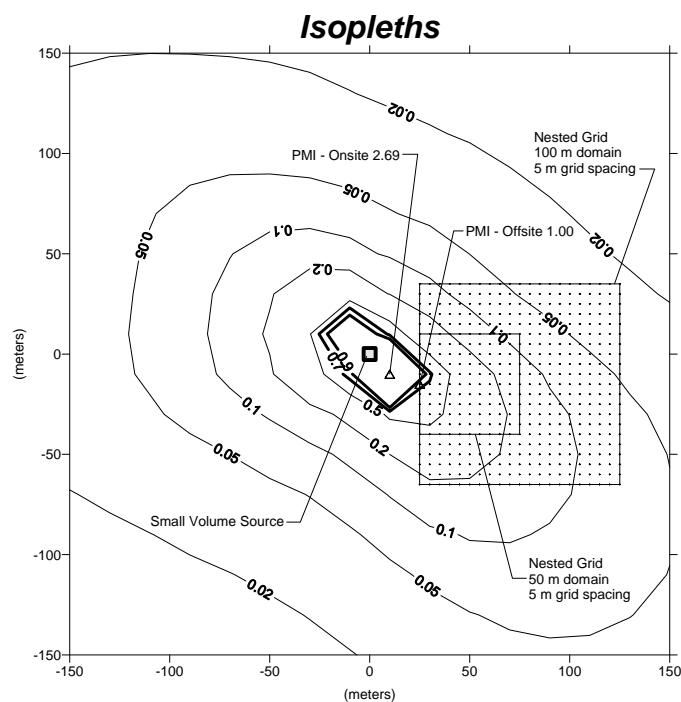


Figure AP3.6.3 – Small Volume Source – Kearny Mesa

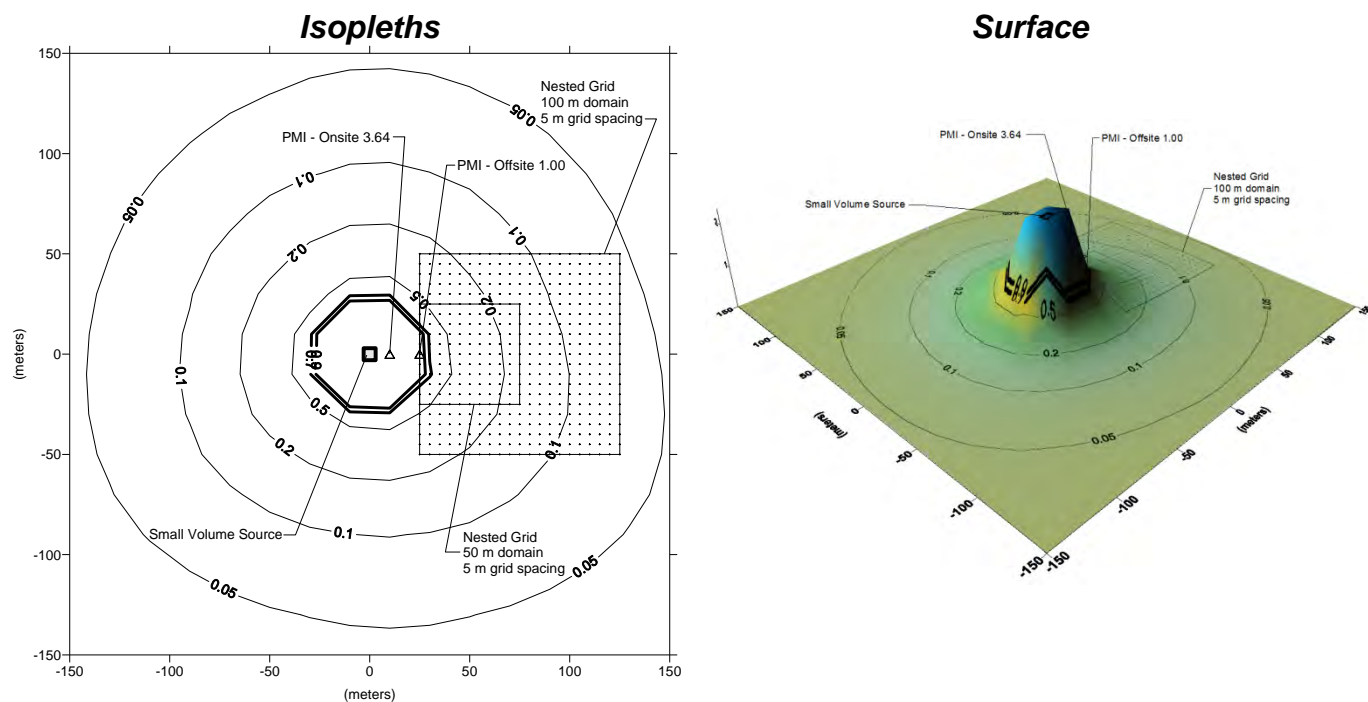


Figure AP3.6.4 – Small Volume Source – Lynnwood

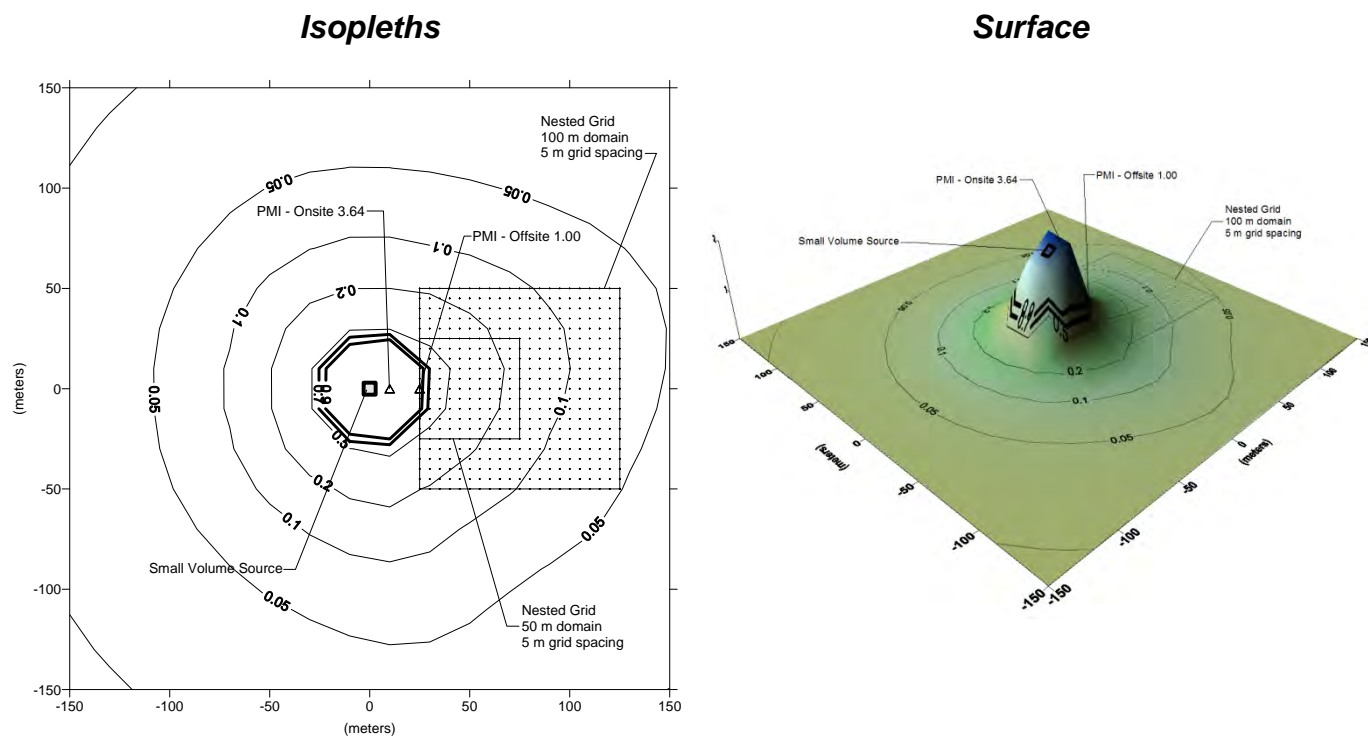


Figure AP3.6.5 – Small Volume Source – San Bernardino

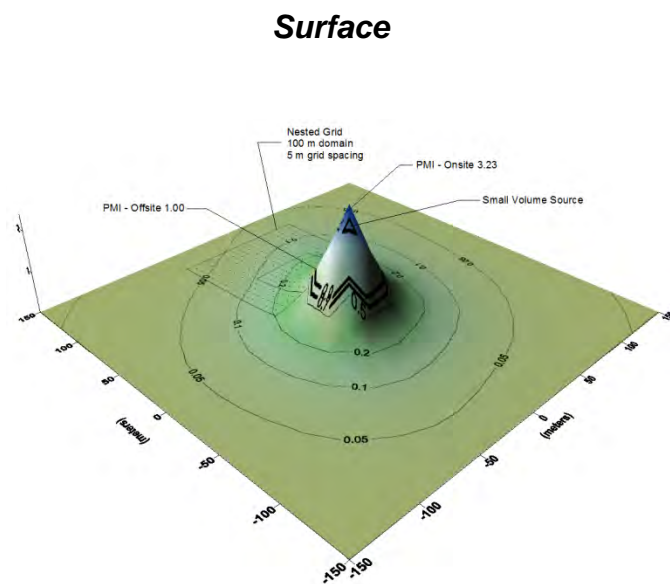
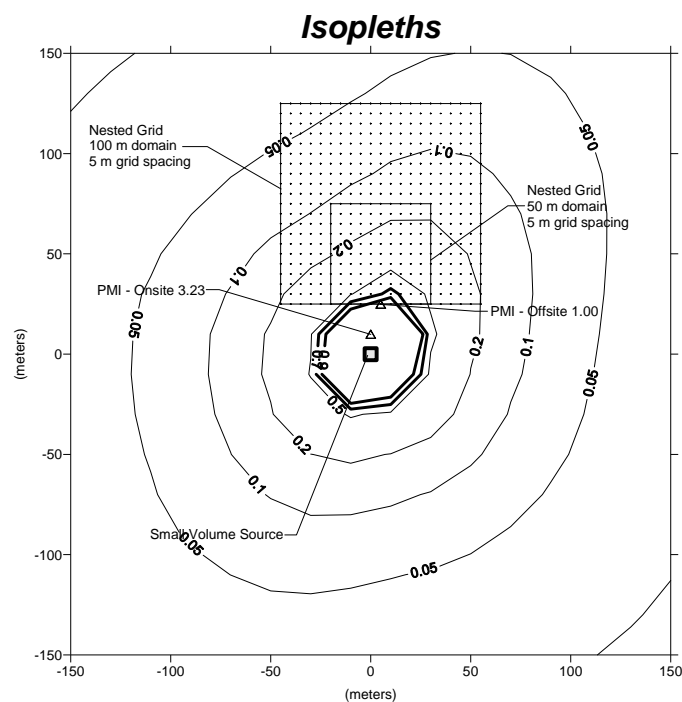


Figure AP3.7.1 – Large Area Source – Costa Mesa

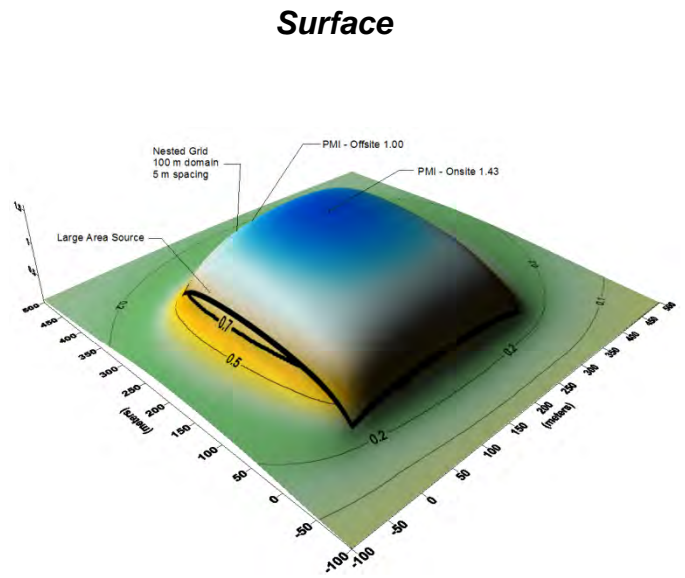
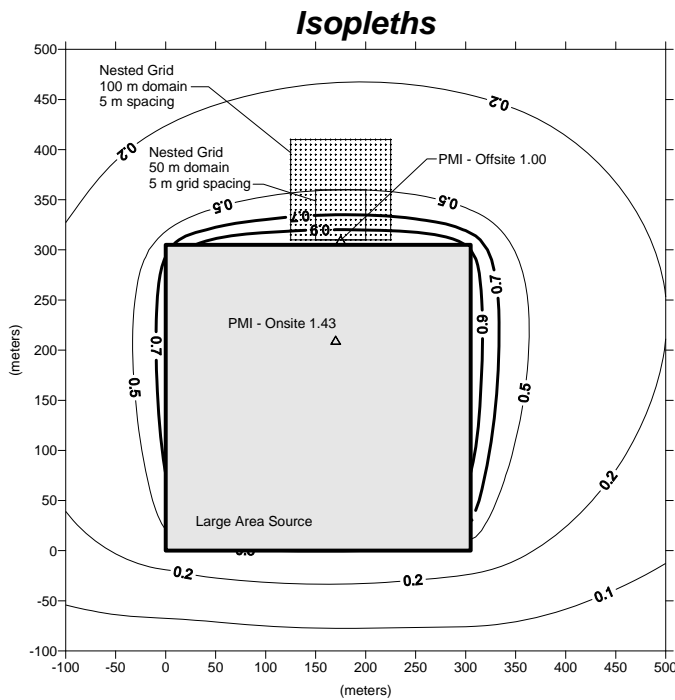


Figure AP3.7.2 – Large Area Source – Fresno Air Terminal

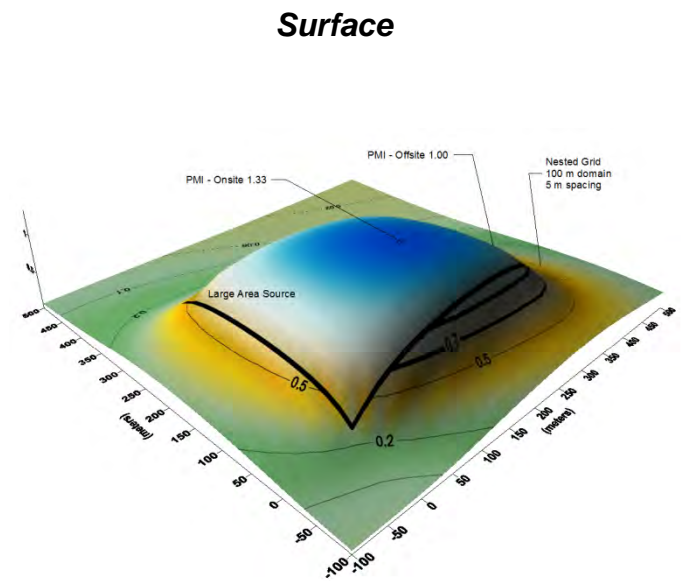
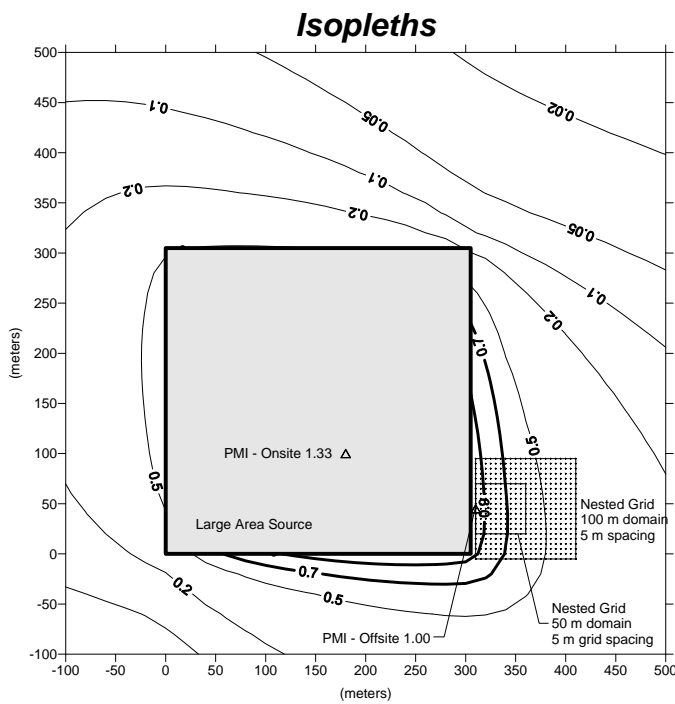


Figure AP3.7.3 – Large Area Source – Kearny Mesa

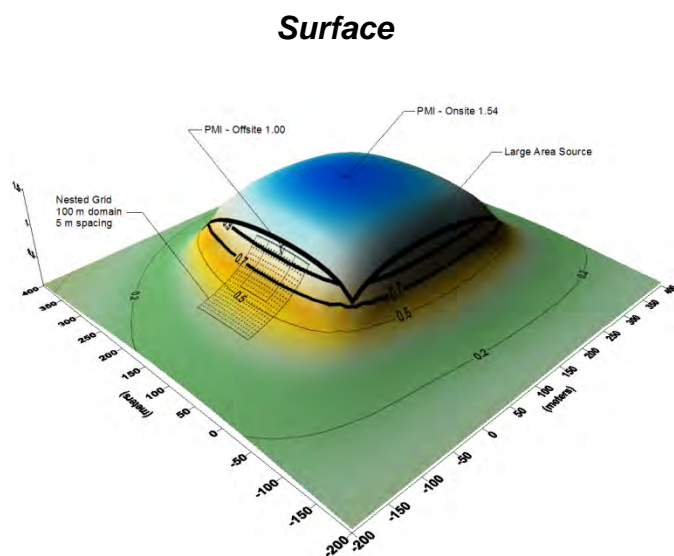
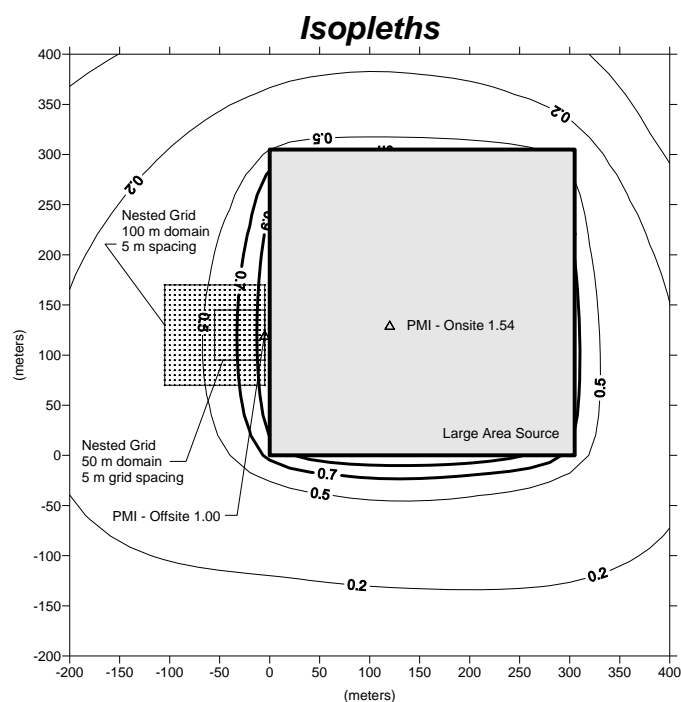


Figure AP3.7.4 – Large Area Source – Lynwood

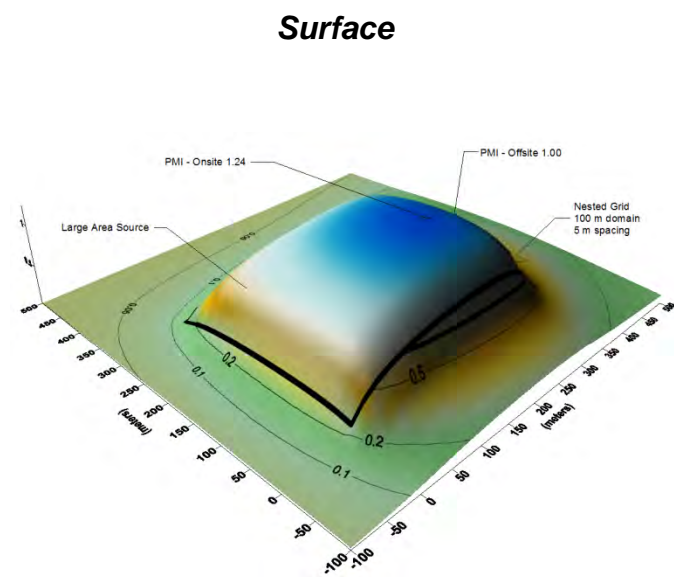
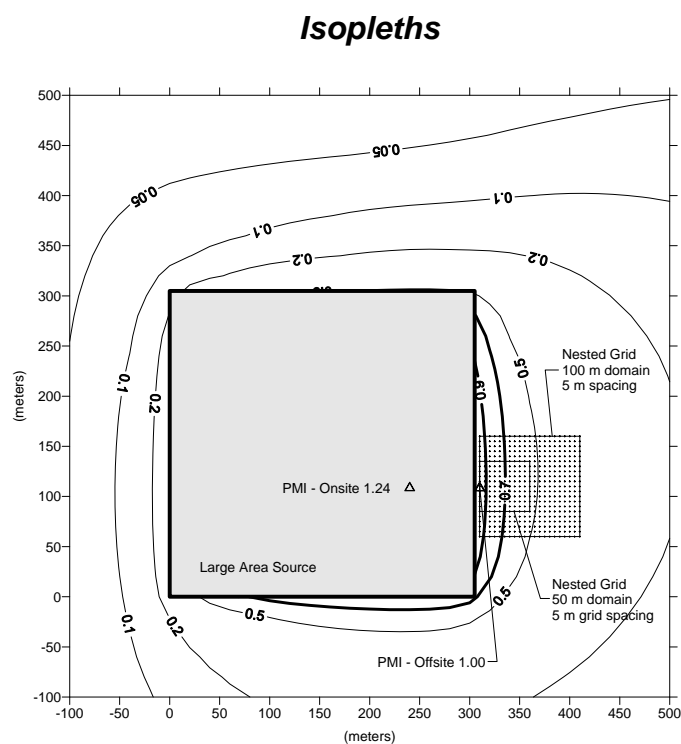


Figure AP3.7.5 – Large Area Source – San Bernardino

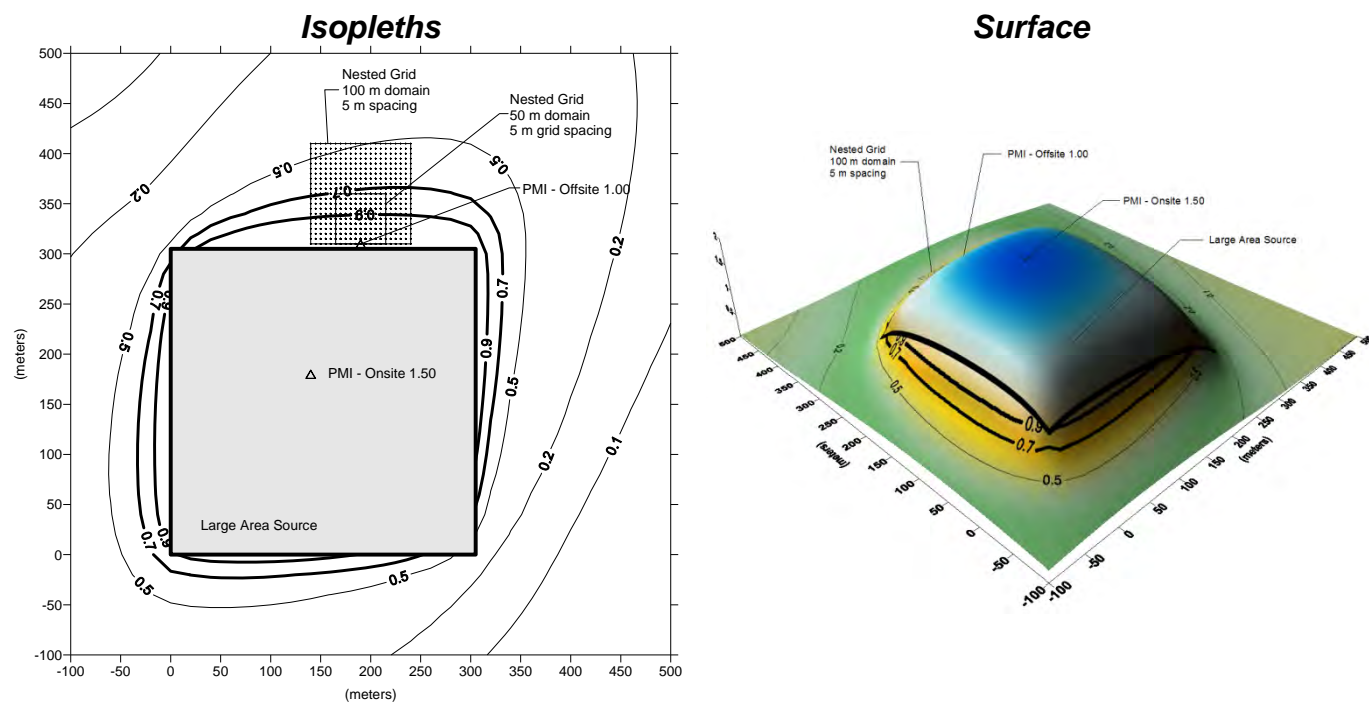


Figure AP3.8.1 – Medium Area Source – Costa Mesa

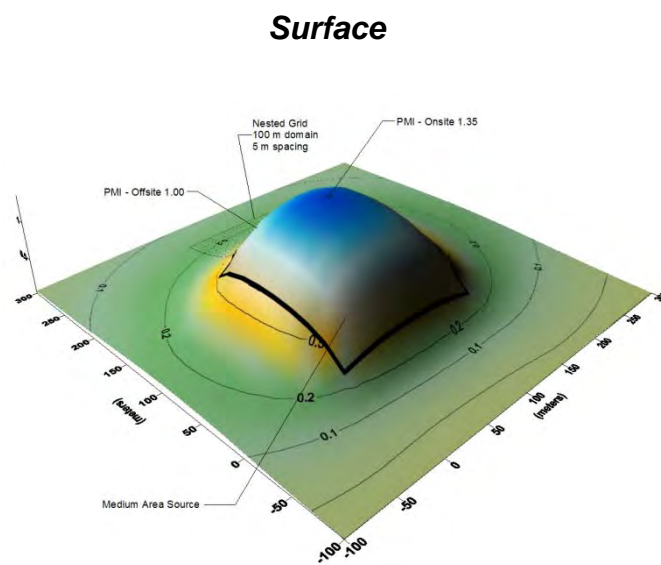
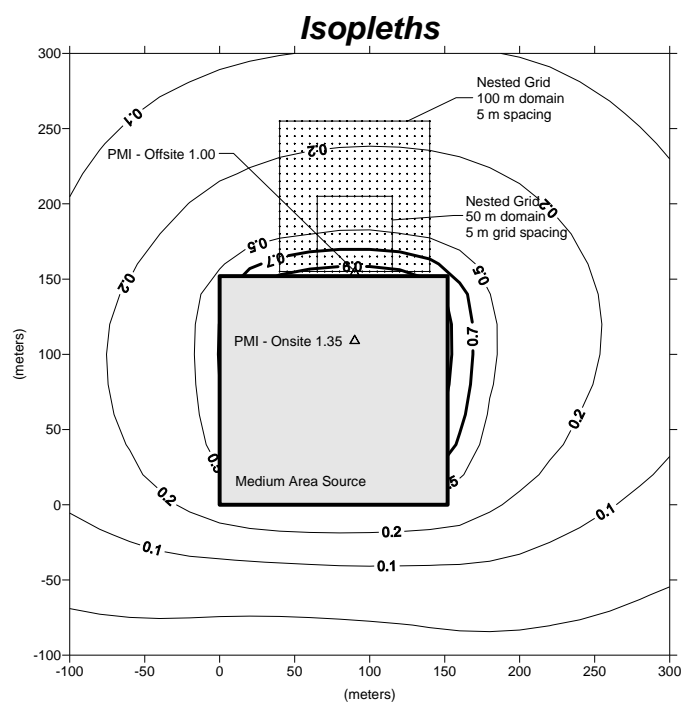


Figure AP3.8.2 – Medium Area Source – Fresno Air Terminal

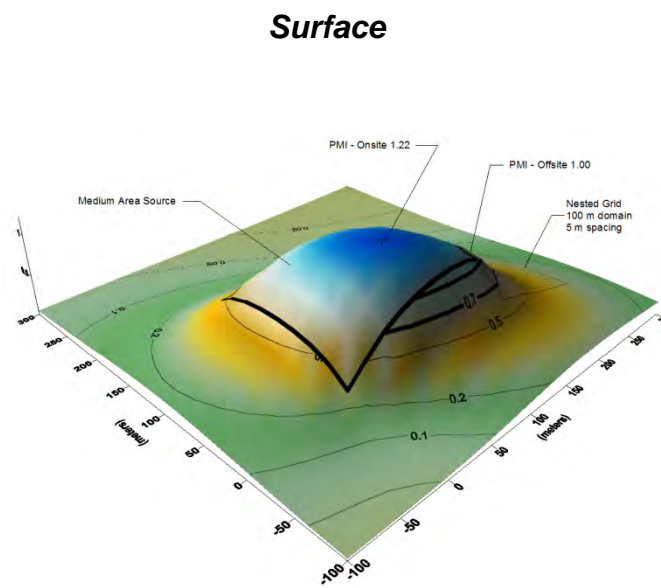
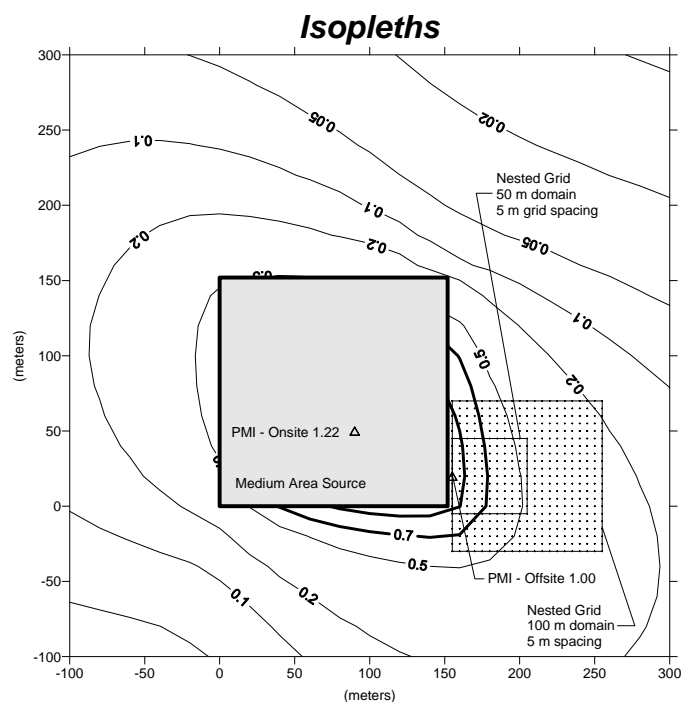


Figure AP3.8.3 – Medium Area Source – Kearny Mesa

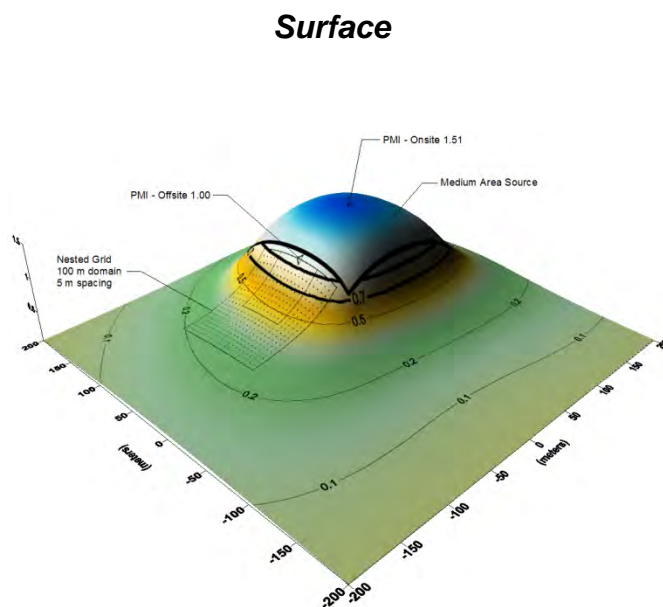
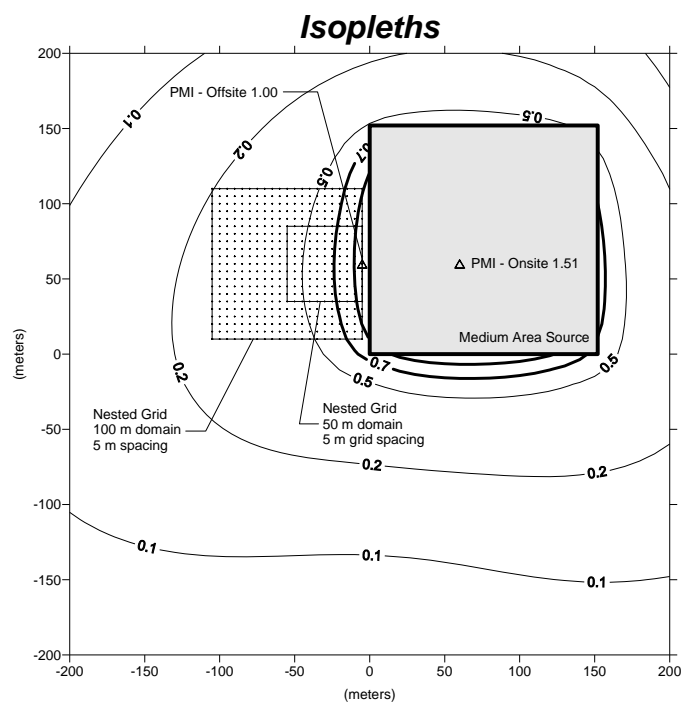


Figure AP3.8.4 – Medium Area Source – Lynwood

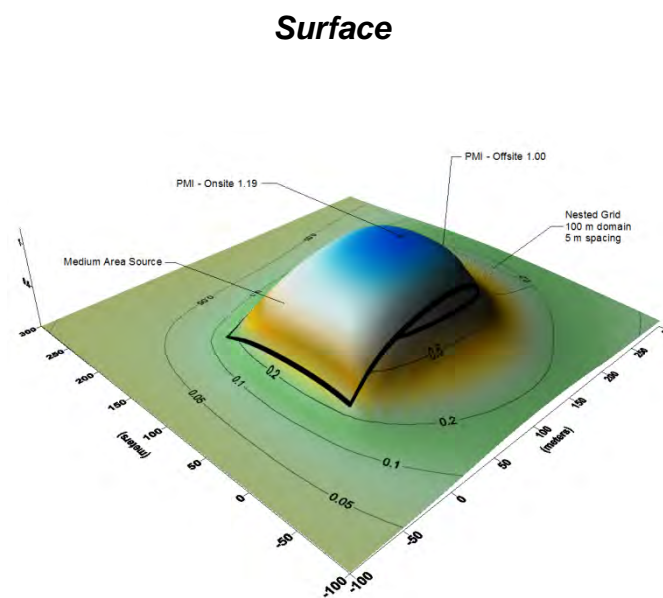
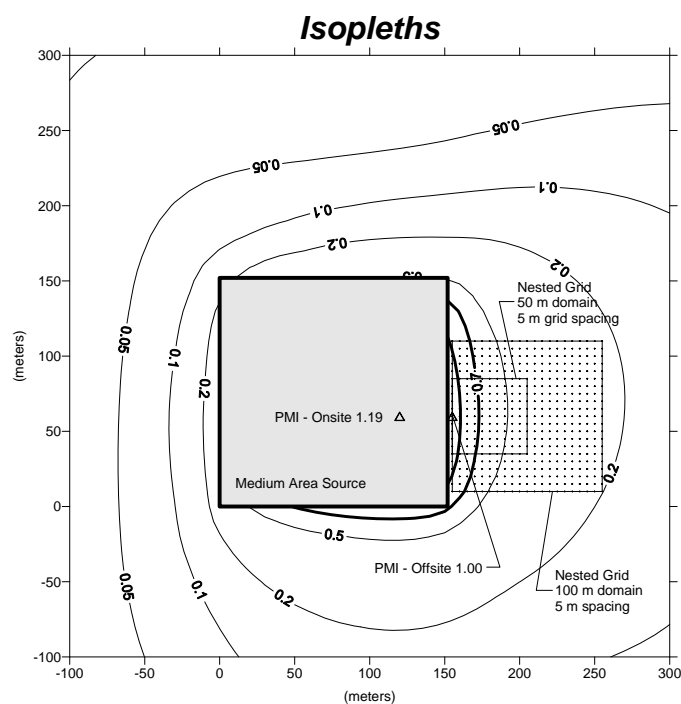


Figure AP3.8.5 – Medium Area Source – San Bernardino

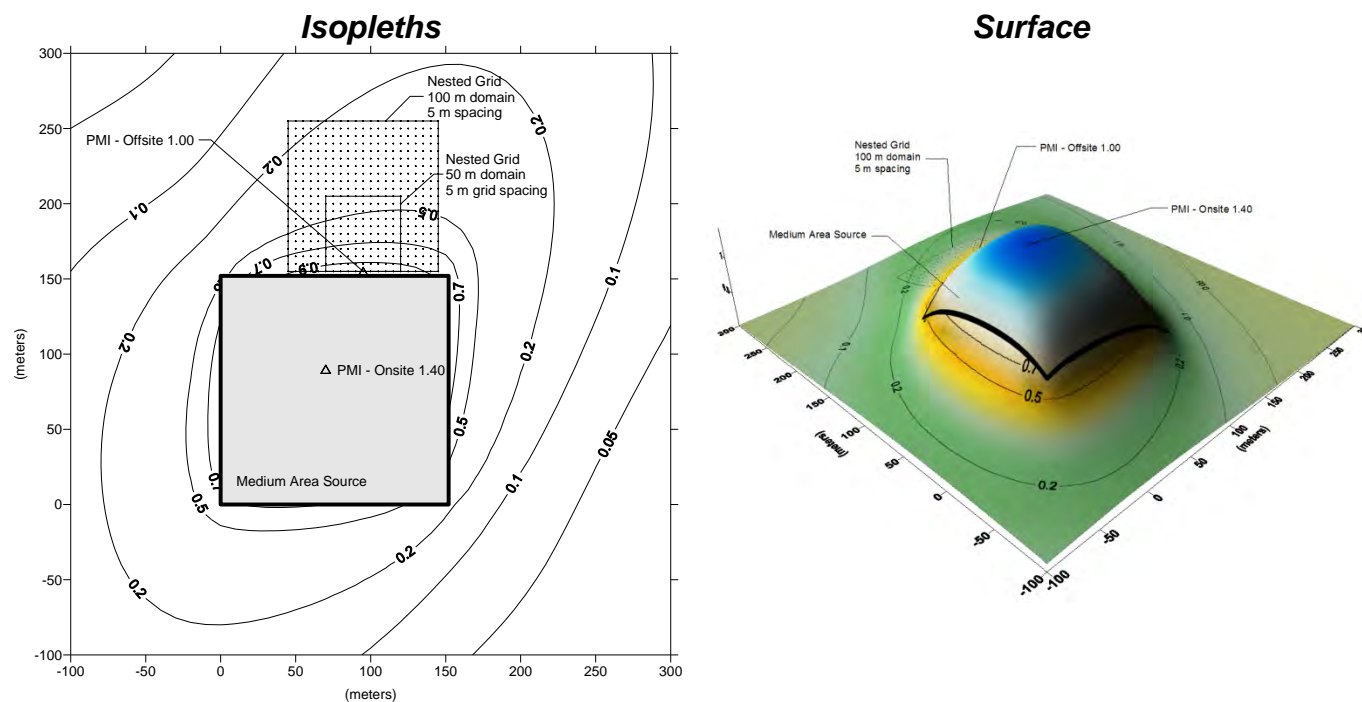


Figure AP3.9.1 – Small Area Source – Costa Mesa

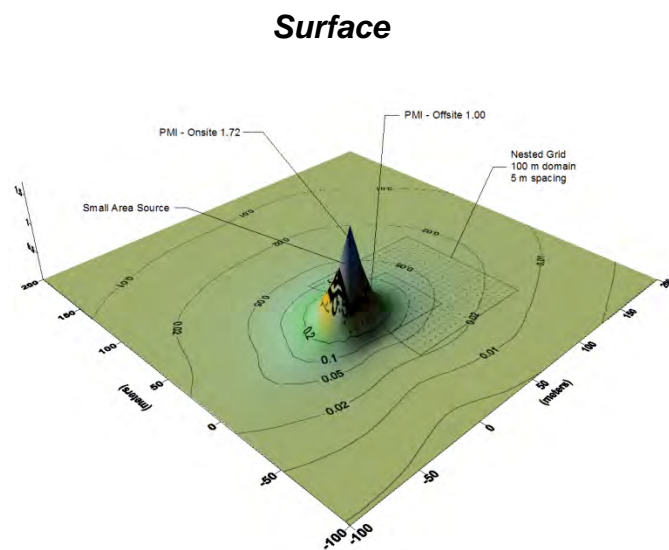
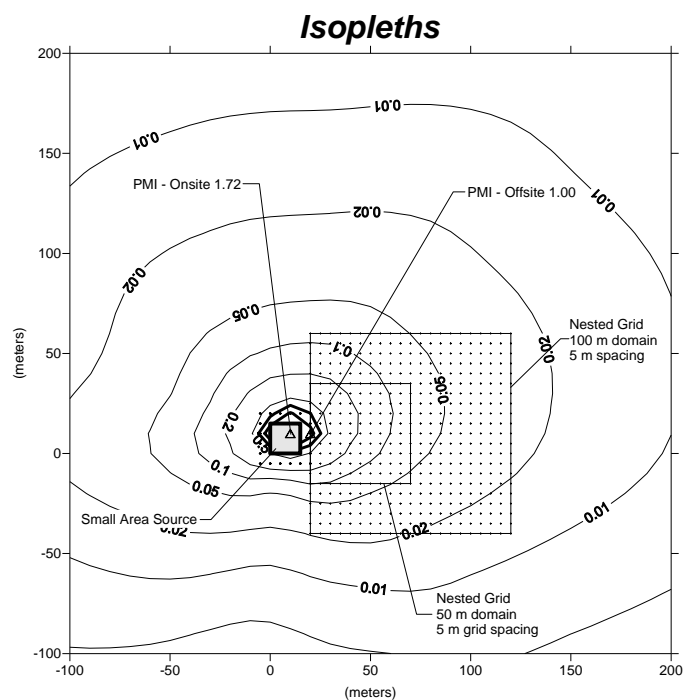


Figure AP3.9.2 – Small Area Source – Fresno Air Terminal

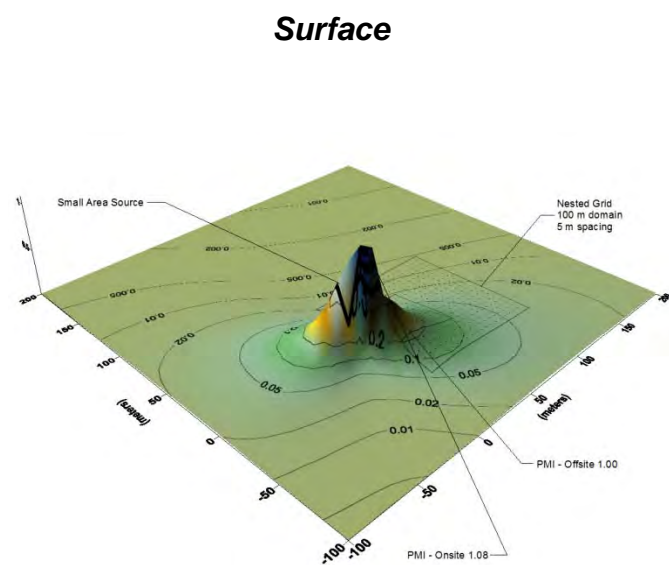
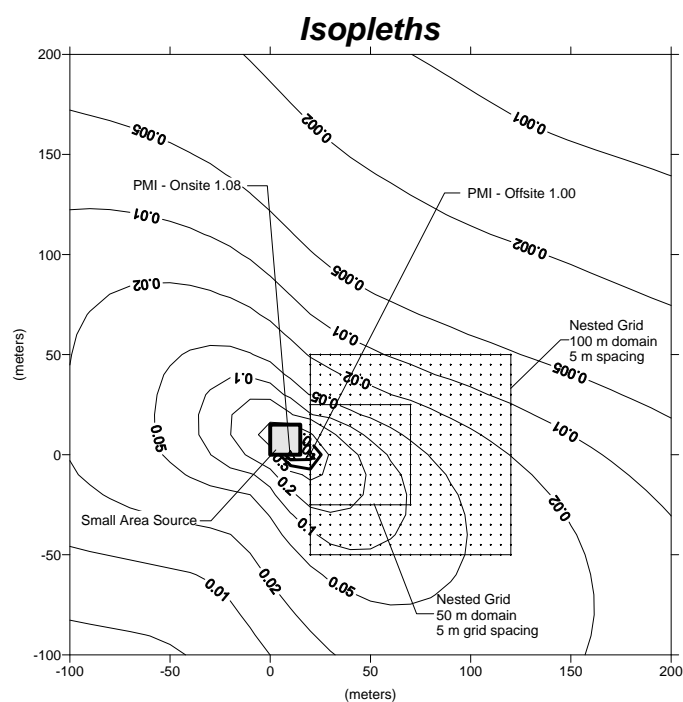


Figure AP3.9.3 – Small Area Source – Kearny Mesa

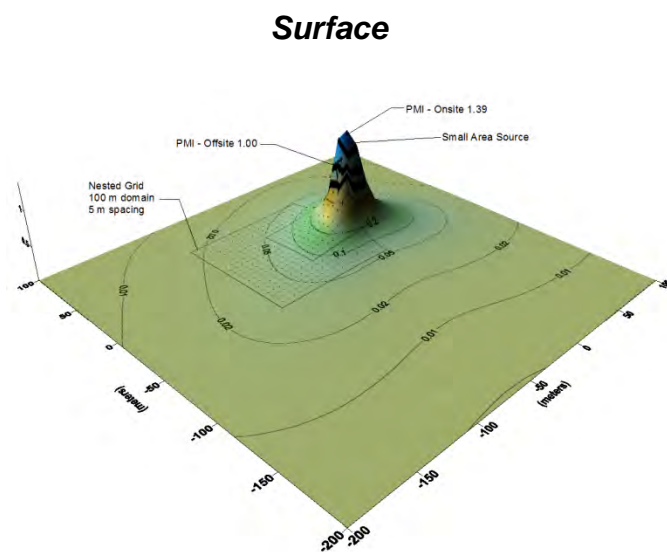
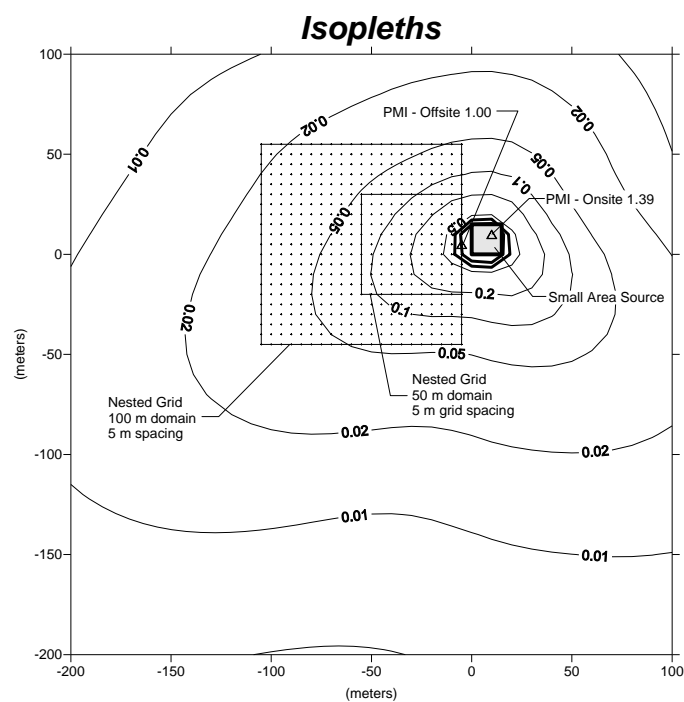


Figure AP3.9.4 – Small Area Source – Lynwood

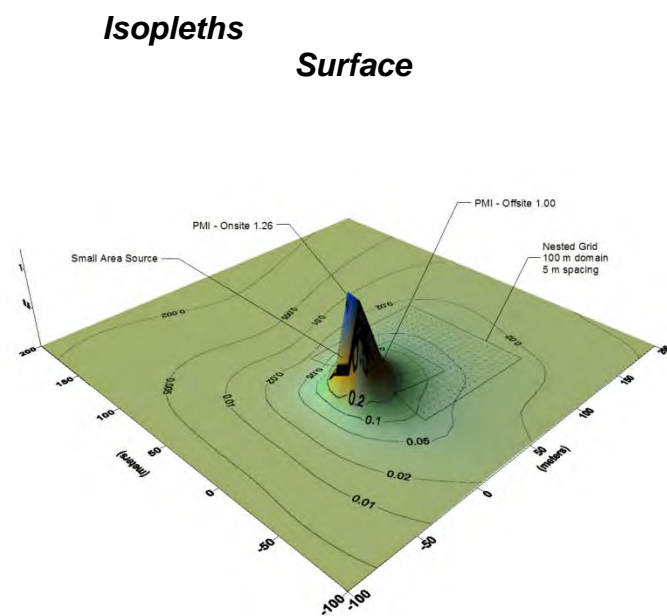
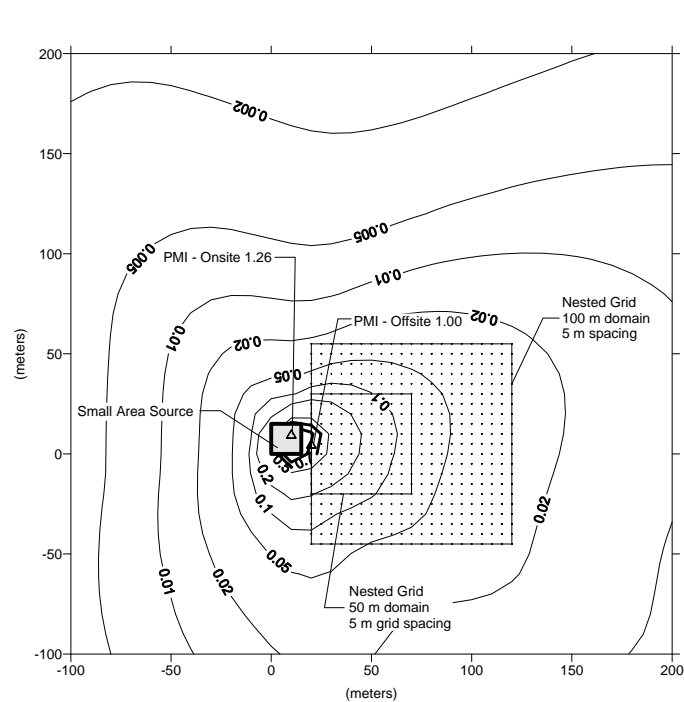


Figure AP3.9.5 – Small Area Source – San Bernardino

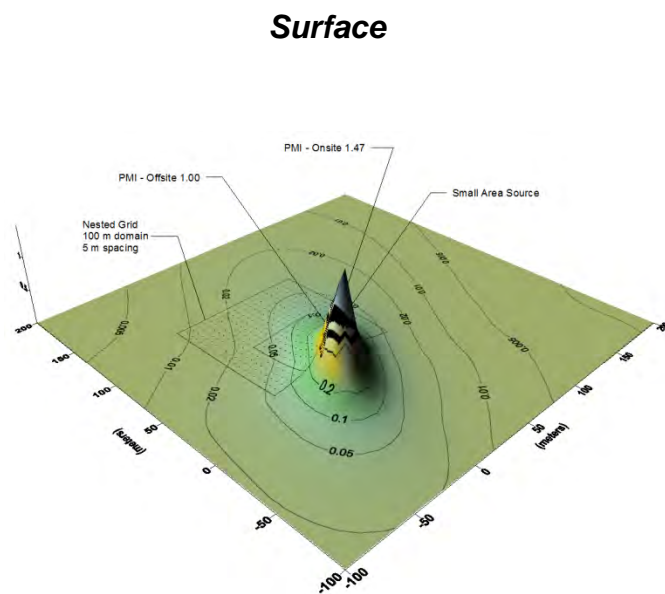
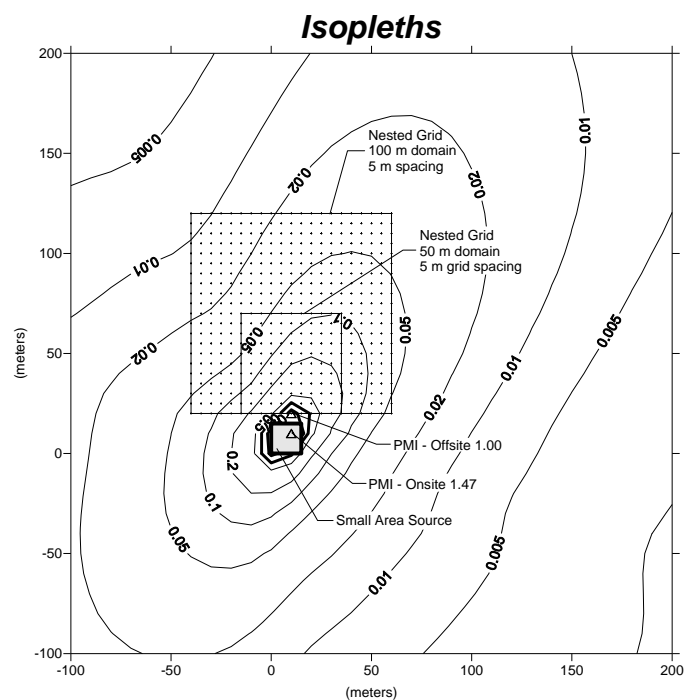


Figure AP3.10.1 – Large Line Source, CALINE – Costa Mesa

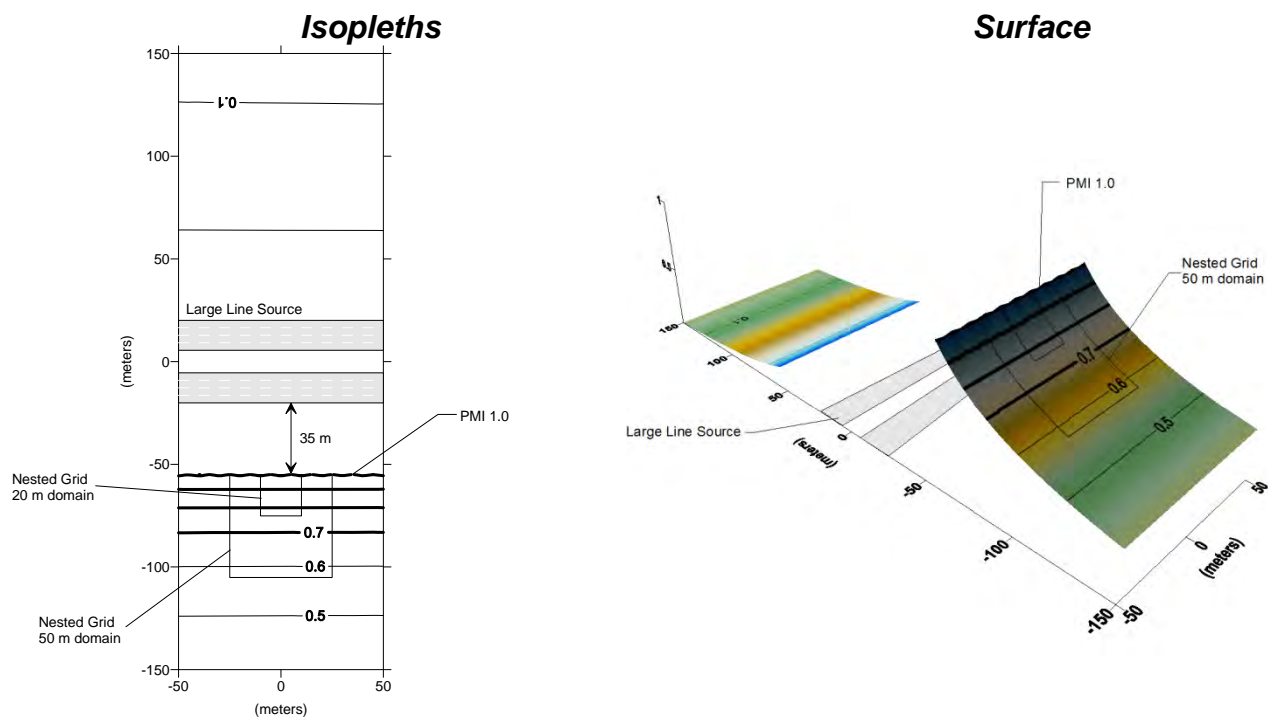


Figure AP3.10.2 – Large Line Source, CALINE – Fresno Air Terminal

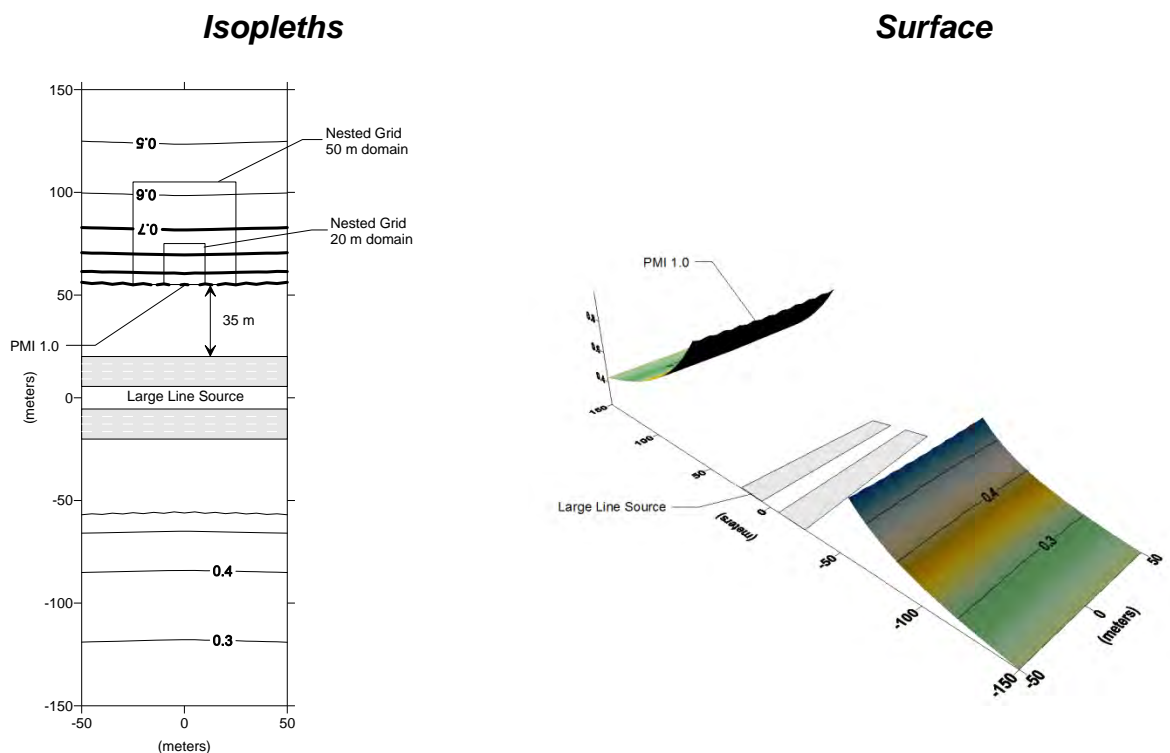


Figure AP3.10.3 – Large Line Source, CALINE – Kearny Mesa

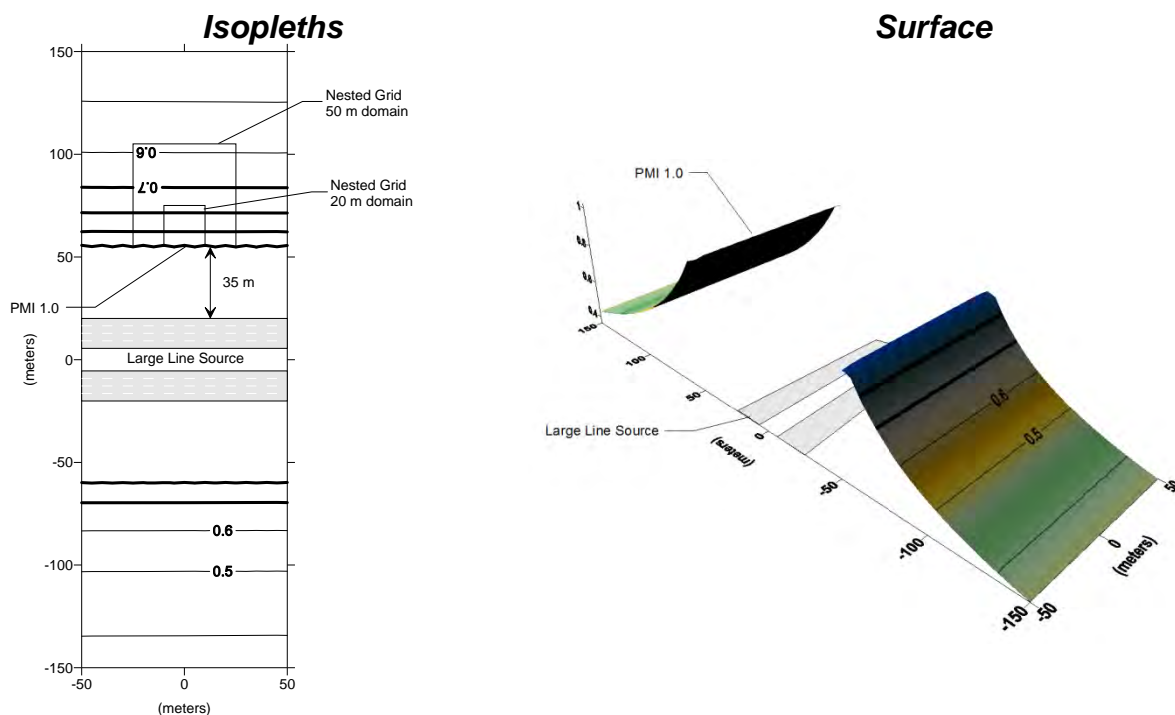


Figure AP3.10.4 – Large Line Source, CALINE – Lynwood

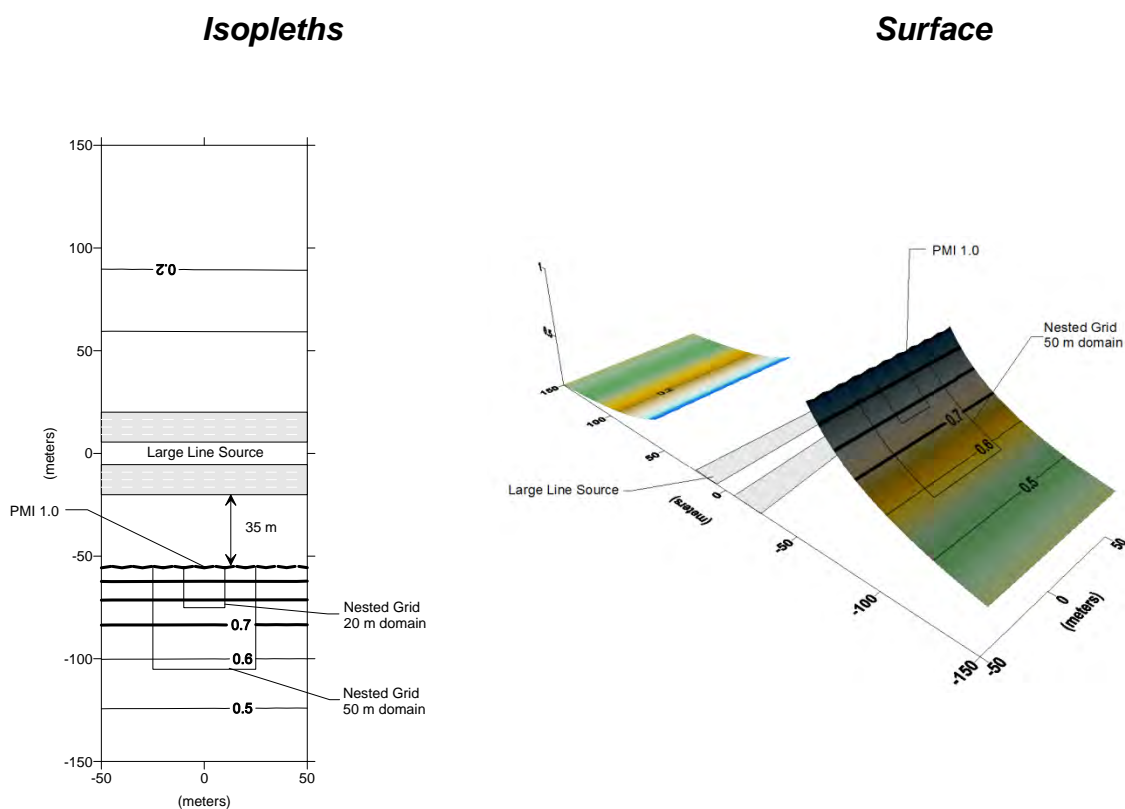


Figure AP3.10.5 – Large Line Source, CALINE – San Bernardino

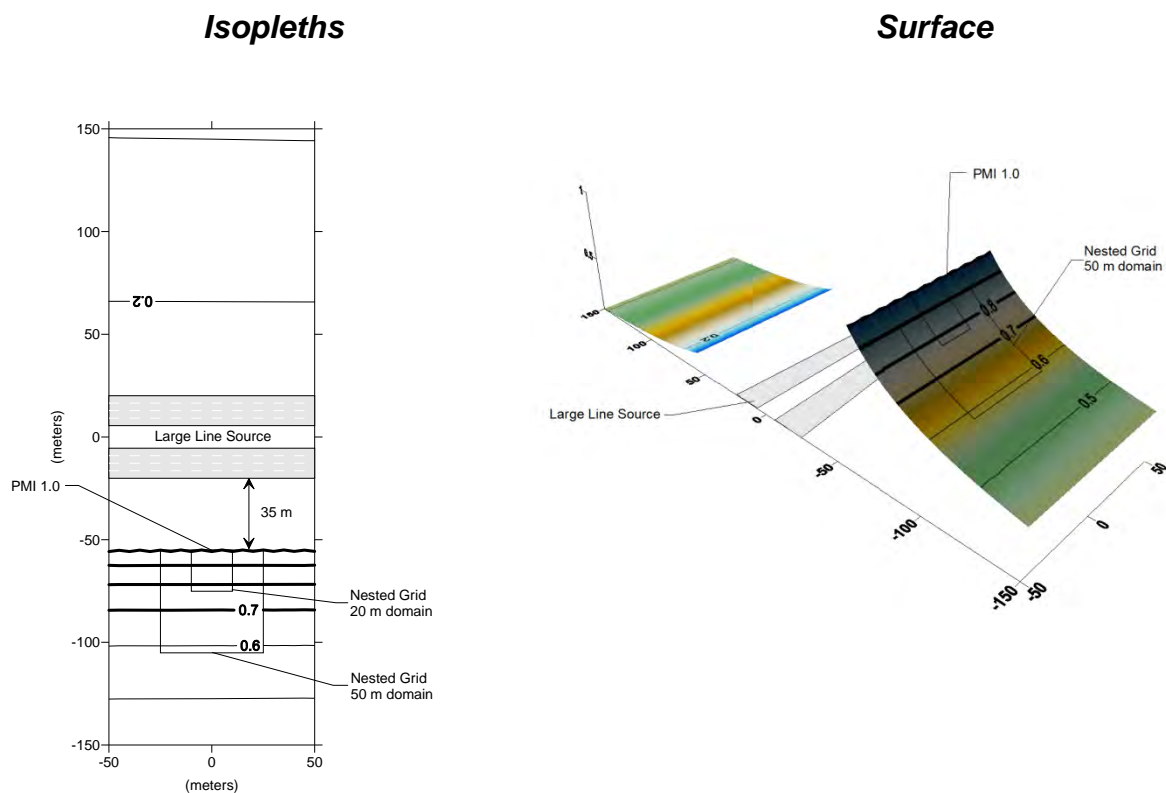


Figure AP3.11.1 – Small Line Source, CALINE – Costa Mesa

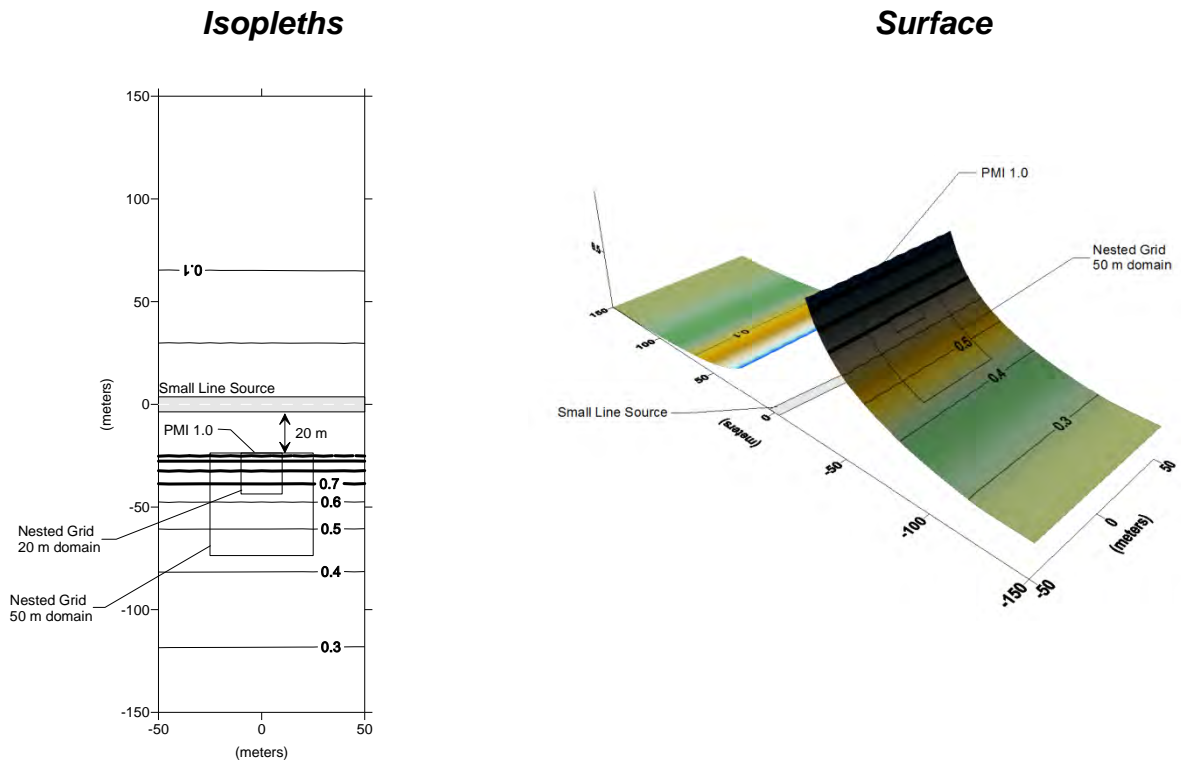


Figure AP3.11.2 – Small Line Source, CALINE – Fresno Air Terminal

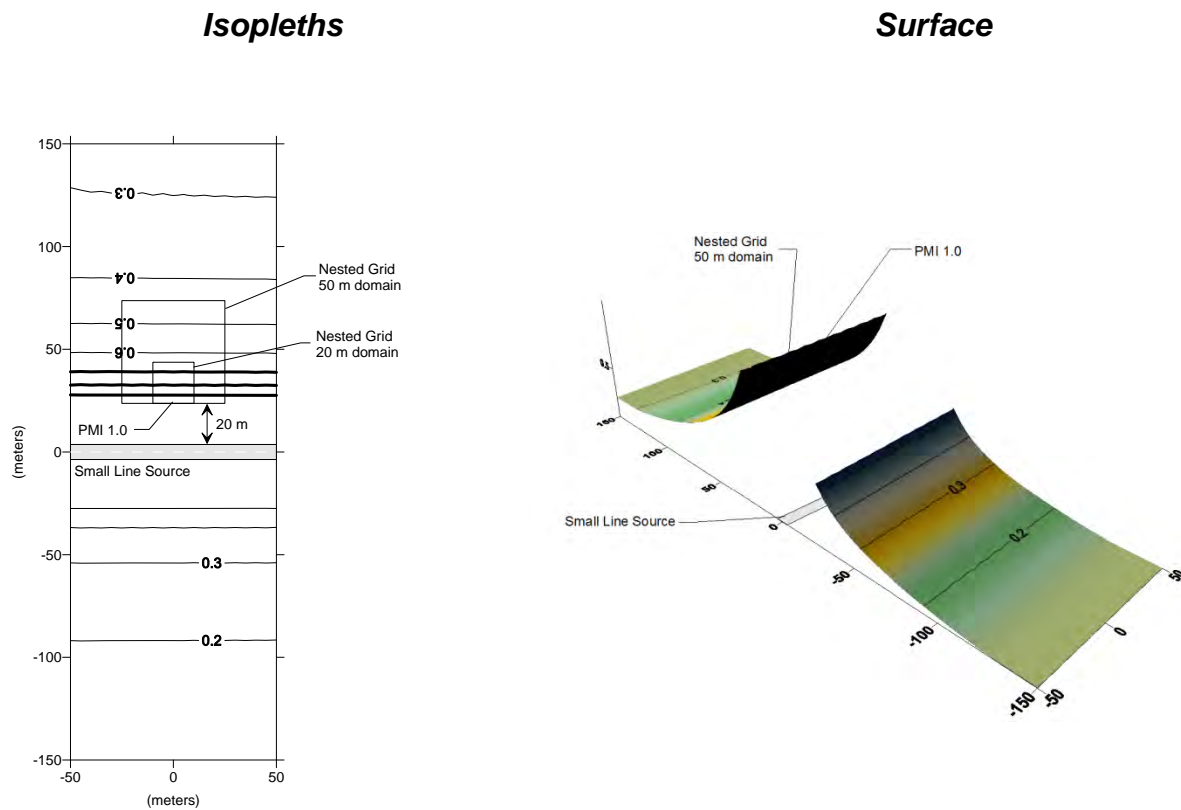


Figure AP3.11.3 – Small Line Source, CALINE – Kearny Mesa

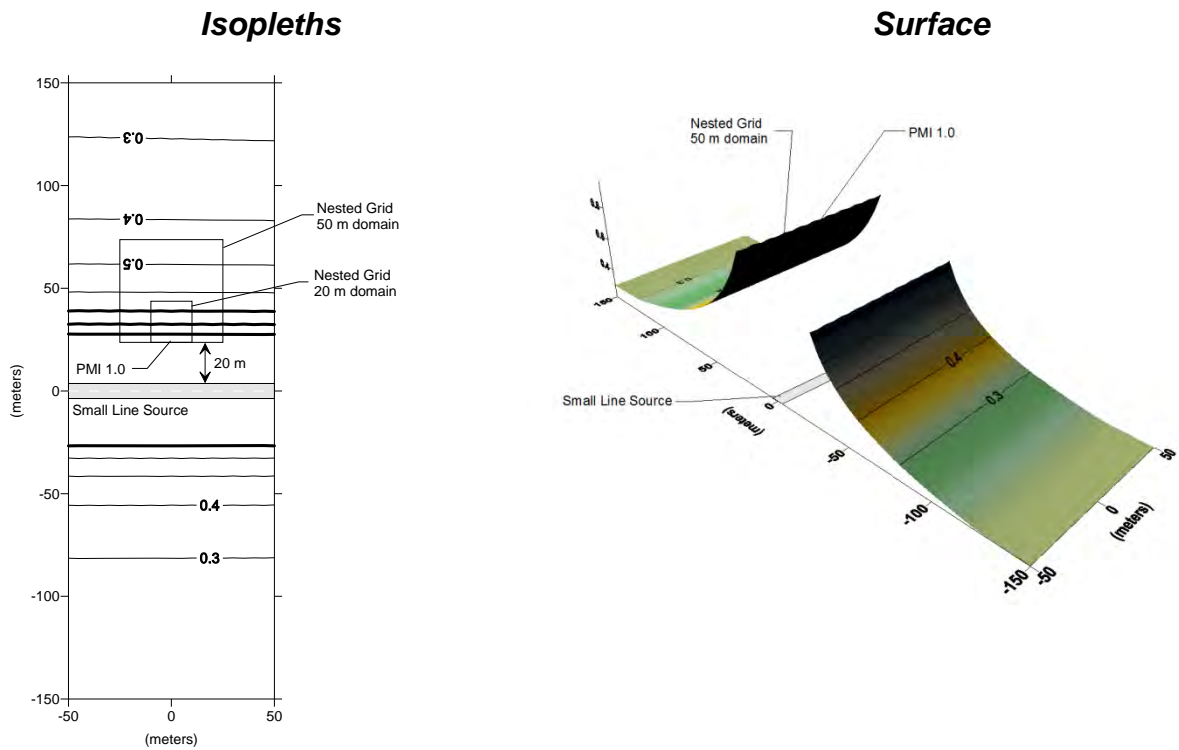


Figure AP3.11.4 – Small Line Source, CALINE – Lynwood

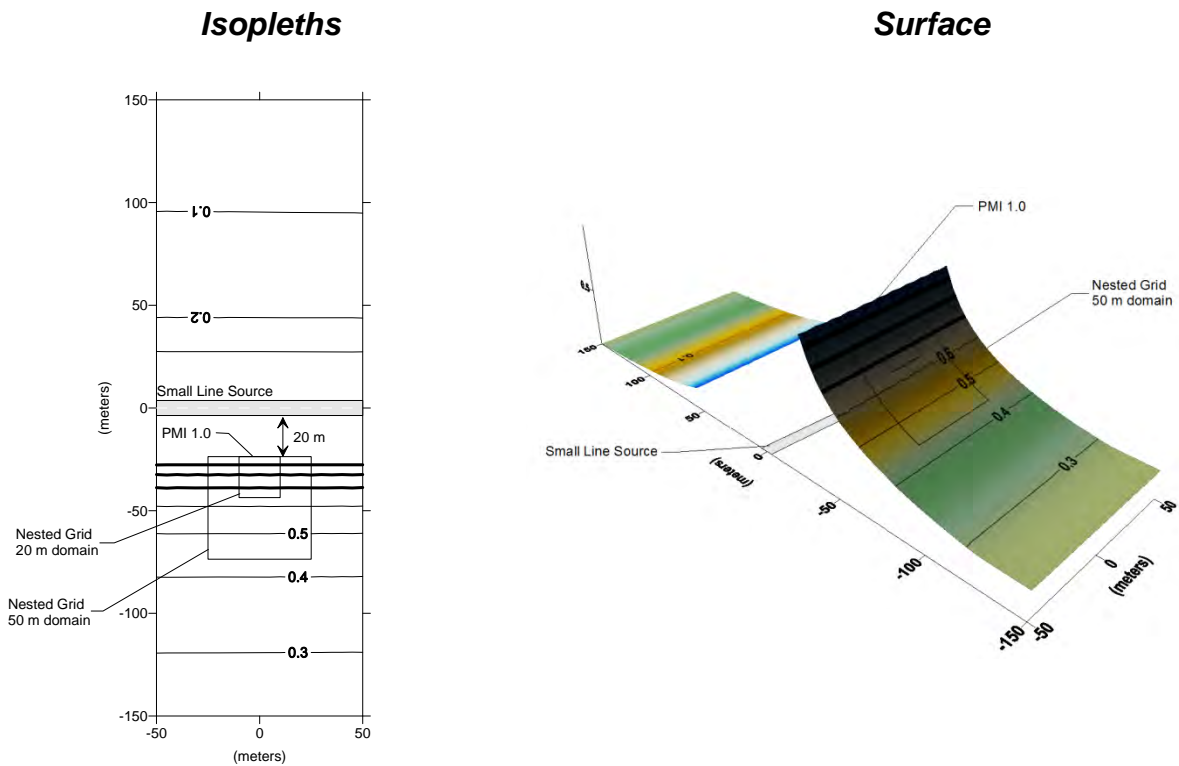
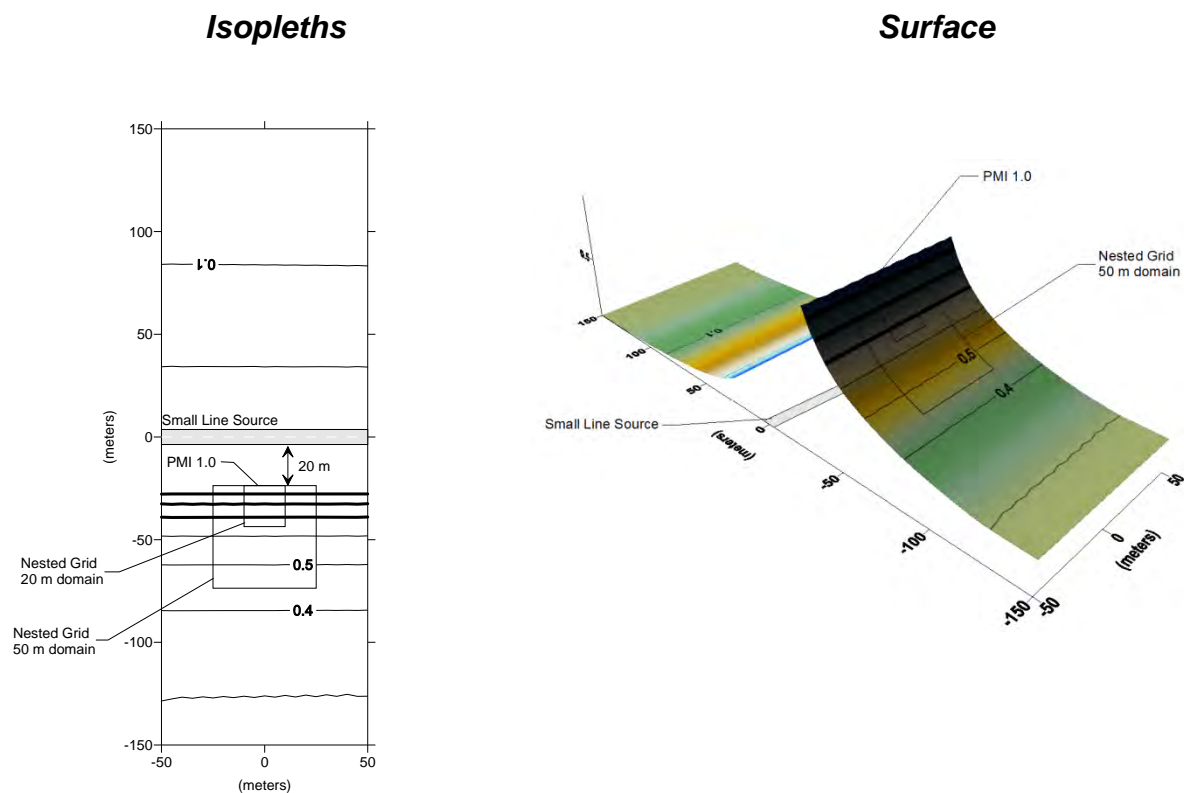


Figure AP3.11.5 – Small Line Source, CALINE – San Bernardino



Appendix 4 – Spatial Average Tables

Table AP4.1.1 – Spatial Average – Point Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.000	1.000	1.000	1.000	1.000
10x10	0.999	0.999	0.999	0.999	0.999
20x20	0.998	0.998	0.997	0.996	0.996
30x30	0.997	0.996	0.994	0.993	0.993
40x40	0.994	0.993	0.990	0.989	0.990
50x50	0.992	0.990	0.985	0.984	0.985
60x60	0.989	0.986	0.979	0.978	0.980
70x70	0.985	0.981	0.972	0.972	0.973
80x80	0.981	0.976	0.965	0.965	0.967
90x90	0.976	0.970	0.956	0.957	0.959
100x100	0.971	0.964	0.947	0.949	0.951

Table AP4.1.2 – Spatial Average – Point Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	1.00	0.99	0.99	0.99	0.99
20x20	0.99	0.98	0.98	0.98	0.98
30x30	0.98	0.97	0.97	0.97	0.97
40x40	0.97	0.95	0.94	0.95	0.95
50x50	0.95	0.92	0.92	0.92	0.93
60x60	0.93	0.89	0.89	0.89	0.90
70x70	0.91	0.86	0.86	0.86	0.87
80x80	0.89	0.83	0.82	0.83	0.84
90x90	0.87	0.79	0.79	0.80	0.81
100x100	0.84	0.76	0.76	0.76	0.78

Table AP4.1.3 – Spatial Average – Point Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	1.01	0.70	0.83	0.83	0.84
20x20	0.85	0.56	0.69	0.68	0.69
30x30	0.73	0.44	0.58	0.58	0.57
40x40	0.63	0.36	0.50	0.49	0.48
50x50	0.55	0.30	0.44	0.43	0.41
60x60	0.49	0.25	0.39	0.40	0.36
70x70	0.44	0.22	0.34	0.37	0.32
80x80	0.39	0.19	0.31	0.33	0.28
90x90	0.36	0.17	0.28	0.27	0.26
100x100	0.32	0.15	0.25	0.24	0.23

Table AP4.2.1 – Spatial Average – Volume Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.90	0.91	0.91	0.90	0.90
20x20	0.82	0.82	0.82	0.82	0.82
30x30	0.75	0.75	0.75	0.75	0.75
40x40	0.68	0.68	0.69	0.68	0.68
50x50	0.63	0.62	0.64	0.63	0.63
60x60	0.58	0.57	0.59	0.58	0.58
70x70	0.54	0.53	0.55	0.54	0.54
80x80	0.50	0.49	0.51	0.50	0.50
90x90	0.47	0.45	0.48	0.47	0.46
100x100	0.44	0.42	0.45	0.44	0.43

Table AP4.2.2 – Spatial Average – Volume Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.82	0.84	0.83	0.82	0.82
20x20	0.69	0.70	0.70	0.69	0.69
30x30	0.59	0.60	0.60	0.59	0.59
40x40	0.51	0.51	0.53	0.52	0.51
50x50	0.45	0.44	0.46	0.45	0.45
60x60	0.40	0.39	0.41	0.40	0.39
70x70	0.35	0.34	0.37	0.36	0.35
80x80	0.32	0.30	0.34	0.32	0.32
90x90	0.29	0.27	0.31	0.29	0.29
100x100	0.26	0.25	0.28	0.27	0.26

Table AP4.2.3 – Spatial Average – Volume Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.76	0.76	0.76	0.76	0.75
20x20	0.60	0.60	0.61	0.60	0.59
30x30	0.49	0.47	0.50	0.49	0.48
40x40	0.41	0.39	0.42	0.41	0.40
50x50	0.35	0.32	0.36	0.35	0.34
60x60	0.30	0.27	0.32	0.30	0.29
70x70	0.26	0.24	0.28	0.26	0.25
80x80	0.23	0.21	0.25	0.23	0.22
90x90	0.20	0.18	0.22	0.20	0.20
100x100	0.18	0.16	0.20	0.18	0.18

Table AP4.3.1 – Spatial Average – Area Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.90	0.95	0.93	0.93	0.93
20x20	0.83	0.89	0.87	0.86	0.87
30x30	0.76	0.85	0.82	0.81	0.81
40x40	0.71	0.80	0.78	0.76	0.77
50x50	0.66	0.77	0.74	0.72	0.73
60x60	0.62	0.73	0.70	0.69	0.69
70x70	0.59	0.70	0.67	0.66	0.66
80x80	0.56	0.67	0.64	0.63	0.64
90x90	0.53	0.64	0.62	0.60	0.61
100x100	0.51	0.62	0.59	0.58	0.59

Table AP4.3.2 – Spatial Average – Area Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1	1	1	1	1
10x10	0.88	0.94	0.91	0.91	0.91
20x20	0.78	0.87	0.83	0.82	0.83
30x30	0.69	0.81	0.76	0.75	0.76
40x40	0.63	0.75	0.70	0.69	0.70
50x50	0.57	0.69	0.65	0.64	0.65
60x60	0.53	0.65	0.61	0.60	0.61
70x70	0.49	0.60	0.57	0.56	0.57
80x80	0.45	0.56	0.54	0.52	0.53
90x90	0.42	0.53	0.50	0.49	0.50
100x100	0.39	0.49	0.47	0.46	0.47

Table AP4.3.3 – Spatial Average – Area Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.64	0.65	0.65	0.65	0.65
20x20	0.44	0.44	0.46	0.46	0.45
30x30	0.32	0.32	0.34	0.26	0.33
40x40	0.25	0.24	0.26	0.21	0.25
50x50	0.20	0.19	0.21	0.17	0.20
60x60	0.16	0.16	0.17	0.14	0.16
70x70	0.13	0.13	0.14	0.14	0.14
80x80	0.11	0.11	0.12	0.12	0.12
90x90	0.10	0.10	0.11	0.10	0.10
100x100	0.09	0.08	0.09	0.09	0.09

Table AP4.4.1 – Spatial Average – Line Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.93	0.93	0.93	0.93	0.93
20x20	0.87	0.88	0.88	0.87	0.88
30x30	0.83	0.83	0.83	0.83	0.83
40x40	0.78	0.79	0.79	0.79	0.79
50x50	0.75	0.75	0.75	0.75	0.75
60x60	0.72	0.72	0.72	0.72	0.72
70x70	0.69	0.70	0.69	0.69	0.70
80x80	0.66	0.67	0.67	0.66	0.67
90x90	0.64	0.65	0.64	0.64	0.65
100x100	0.62	0.63	0.62	0.62	0.63

Table AP4.4.2 – Spatial Average – Line Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.88	0.88	0.88	0.88	0.88
20x20	0.80	0.80	0.79	0.80	0.80
30x30	0.73	0.74	0.73	0.73	0.73
40x40	0.68	0.69	0.67	0.68	0.68
50x50	0.64	0.64	0.63	0.64	0.64
60x60	0.60	0.61	0.59	0.60	0.61
70x70	0.57	0.58	0.56	0.57	0.58
80x80	0.54	0.55	0.54	0.54	0.55
90x90	0.52	0.53	0.51	0.52	0.53
100x100	0.50	0.51	0.49	0.50	0.51

Appendix 5 – Tilted Spatial Averaging

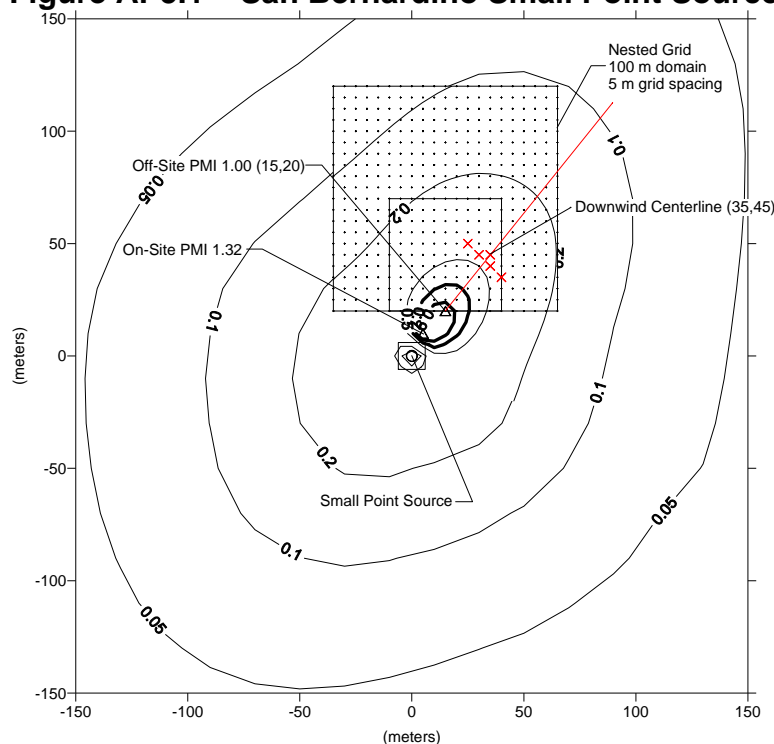
Tilted Spatial Averaging

Small sources tend to show an offsite PMI located at the fence line. It may be necessary to tilt the spatial averaging receptor field when the predominate wind direction carries the average plume centerline askew from the cardinal directions.

The first step in tilting the receptor field is to determine the centerline of the tilted receptor field. The centerline intersects the offsite PMI in the near field. We recommend locating the far end of the centerline by selecting receptors from the 5m spaced grid with the highest concentrations located approximately 30 meters from the offsite PMI.

For example, in the case of San Bernardino meteorology and a small point source, the offsite PMI is located at (15, 20). The dominate plume centerline can be determined from the existing set of receptors spaced at a 5 m grid cell resolution. The maximum concentration located approximately 30 meters from the offsite PMI can be used for the centerline. In this case the plume centerline was determined by plotting the receptors with the five highest concentrations and making a subjective selection of the centerline receptor at (35, 45). See red “x” receptors in Figure E1.

Figure AP5.1 – San Bernardino Small Point Source



Polar coordinates can be easily calculated from the two points, (15, 20) and (35, 45), with basic trigonometry. In this case, $dy/dx = 1.250$, and the centerline tilted angle is 38.660 degrees from vertical (51.340 degrees from horizontal).

$$\tan \theta = \frac{dy}{dx} = \frac{45 - 20}{35 - 15} = \frac{25}{20} = 1.250$$

Therefore, $\theta = 38.660^\circ$

We recommend that the polar receptor field cover half of a circular area, a 180 degree arc. So for our example the polar receptors centered on 38.660 degrees will sweep an arc from 308.660 degrees to 128.660 degrees (i.e., $38.660^\circ \pm 90^\circ$).

Polar receptors in AERMOD are easy to specify. Receptors should be placed on radials incremented every five meters. The polar angle of the radials should be placed to closely represent 5 meter grid spacing. For example, Table E1 below shows the angular increment of radials for receptor placement out to 25m from the offsite PMI.

Table AP5.1 – Recommended Spacing for Tilted Polar Nested Grid

Radial Distance from PMI	0m	5m	10m	15m	20m	25m
Angle Increment (deg)	PMI	60.000	30.000	18.000	13.846	11.250
Resultant spacing along arc	PMI	5.24m	5.24m	4.71m	4.83m	4.91m

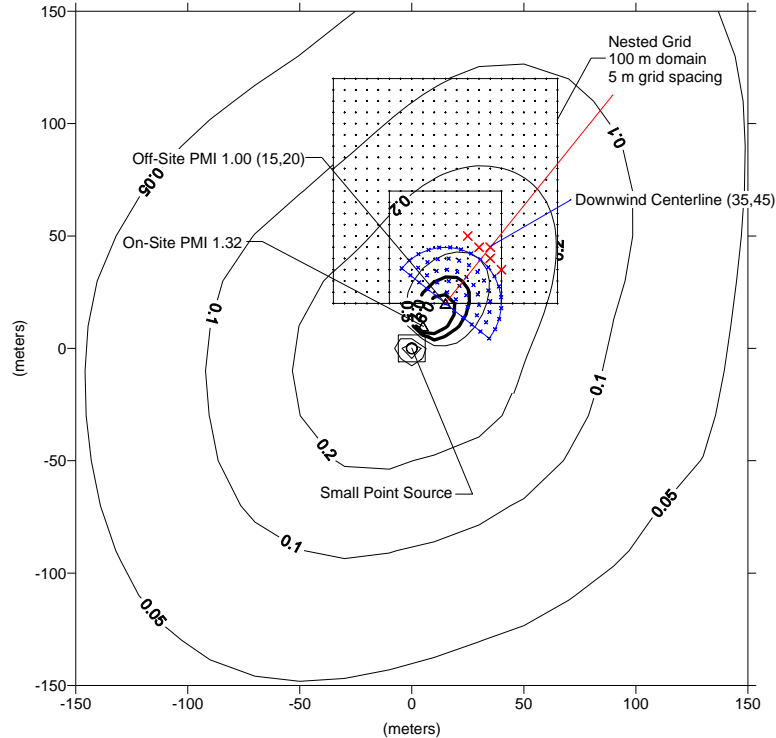
As a result of the above receptor spacing, the following field of polar receptors in Table E2 is needed for the San Bernardino example.

Table AP5.2 – Tilted Nested Grid for San Bernardino Example

Radial Distance →	5m	10m	15m	20m	25m
Radial Direction (degrees)					
1	308.660	308.660	308.660	308.660	308.660
2	8.660	338.660	326.660	322.506	319.910
3	68.660	8.660	344.660	336.352	331.160
4	128.660	38.660	2.660	350.198	342.410
5	-	68.660	20.660	4.044	353.660
6	-	98.660	38.660	17.891	4.910
7	-	128.660	56.660	31.737	16.160
8	-	-	74.660	45.583	27.410
9	-	-	92.660	59.429	38.660
10	-	-	110.660	73.275	49.910
11	-	-	128.660	87.121	61.160
12	-	-	-	100.968	72.410
13	-	-	-	114.814	83.660
14	-	-	-	128.660	94.910
15	-	-	-	-	106.160
16	-	-	-	-	117.410
17	-	-	-	-	128.660
Note: Be sure to include the offsite PMI in the polar spatial average.					

Figure E2 shows the resulting receptors for the above field as blue “x”s.

Figure AP5.2 – Tilted Nested Polar Grid for San Bernardino Point – Small



As an alternative, a rectangular tilted receptor field can also be created as shown in Figure E3.1, below. The tilted rectangular field shown below requires more calculations than the tilted polar field above because discrete receptors must be generated outside of AERMOD. We recommend the tilted polar field approach because of the simplicity of inputting polar receptors into AERMOD.

Table E3.1 shows a summary of the spatial averaging of tilted nested grids for the San Bernardino meteorological data. In this example, there is little difference between the regular rectangular grid and the tilted rectangular grid.

Figures E3.2 and E3.3 show the tilted grids for the volume and area sources examples. In these cases, the tilted grid spatial average is higher than the non-tilted grid. Table E3.2 shows the spatial average increases from 0.59 to 0.69 for the 20m x 20m nested grid.

Figures E4.1-4.3 show similar trends for nested grids, in this case with meteorological data from the Fresno Air Terminal.

Figure AP5.3.1 – Tilted Nested Rectangular Grid for San Bernardino Point – Small

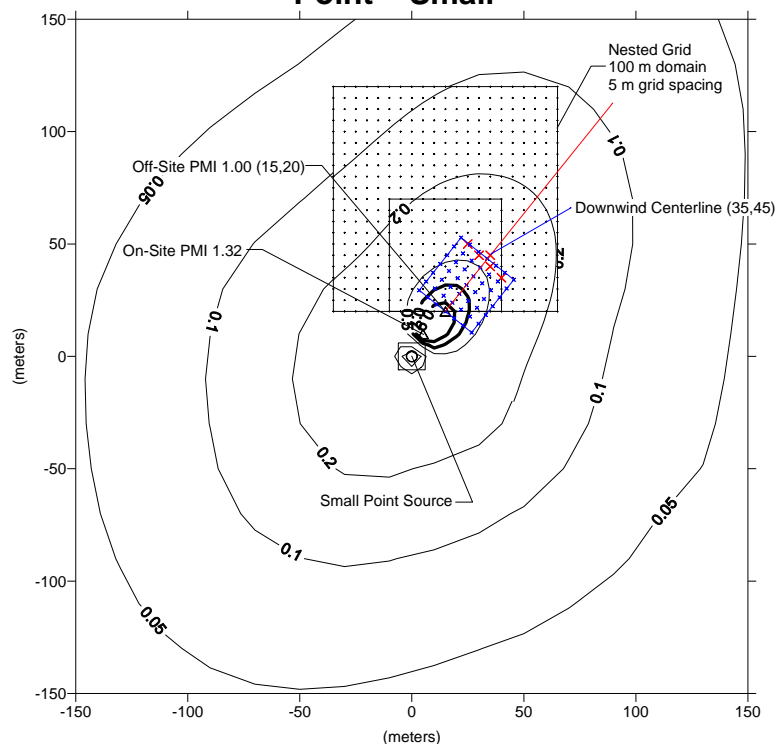


Table AP5.3.1 – Spatial Average – San Bernardino – Small Point Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.91	Polar, R = 5m
100	0.84	0.84	-	Rectangular, 10m x 10m
157	-	-	0.81	Polar, R = 10m
353	-	-	0.71	Polar, R = 15m
400	0.69	0.68	-	Rectangular, 20m x 20m
628	-	-	0.63	Polar, R = 20m
900	0.57	0.58	-	Rectangular, 30m x 30m
982	-	-	0.56	Polar, R = 25m

**Figure AP5.3.2 – Tilted Nested Grid for San Bernardino
Volume – Small**

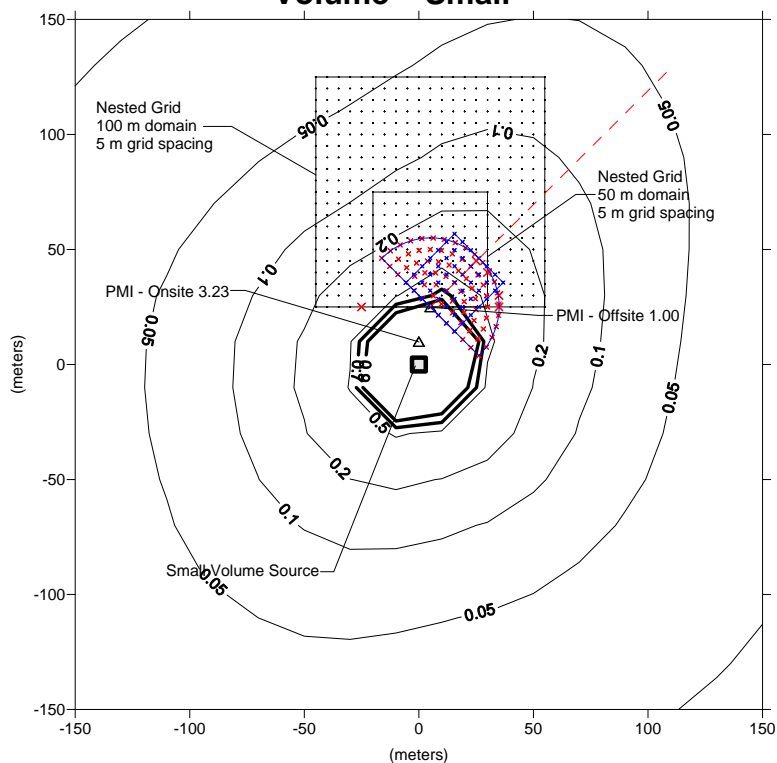


Table AP5.3.2 – Spatial Average – San Bernardino – Small Volume Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.94	Polar, R = 5m
100	0.75	0.83	-	Rectangular, 10m x 10m
157	-	-	0.86	Polar, R = 10m
353	-	-	0.77	Polar, R = 15m
400	0.59	0.69	-	Rectangular, 20m x 20m
628	-	-	0.68	Polar, R = 20m
900	0.48	0.57	-	Rectangular, 30m x 30m
982	-	-	0.56	Polar, R = 25m

Figure AP5.3.3 – Tilted Nested Grid for San Bernardino Area – Small

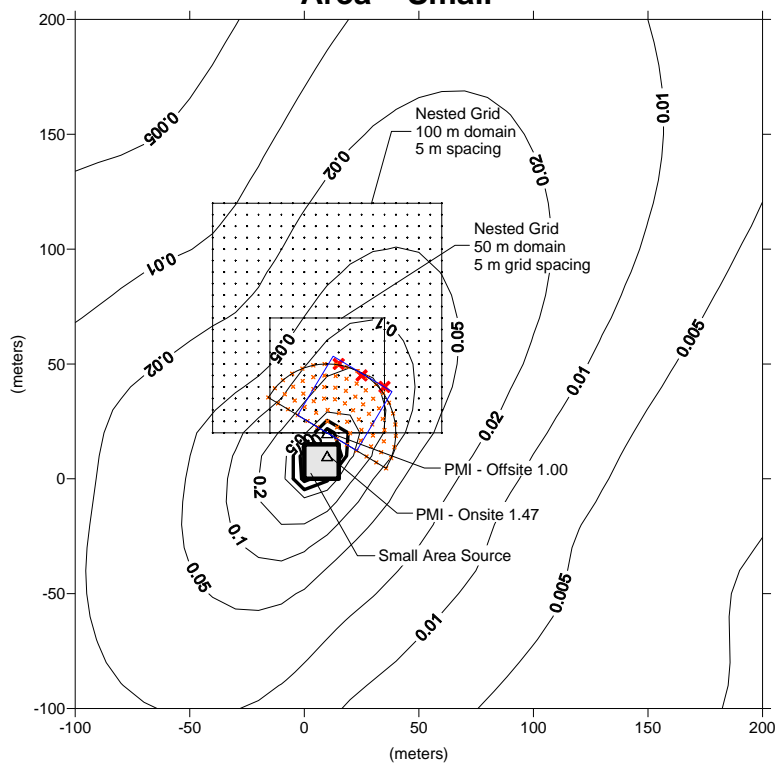


Table AP5.3.3 – Spatial Average – San Bernardino – Small Area Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.86	Polar, R = 5m
100	0.65	0.71	-	Rectangular, 10m x 10m
157	-	-	0.68	Polar, R = 10m
353	-	-	0.52	Polar, R = 15m
400	0.45	0.50	-	Rectangular, 20m x 20m
628	-	-	0.42	Polar, R = 20m
900	0.33	0.36	-	Rectangular, 30m x 30m
982	-	-	0.34	Polar, R = 25m

Figure AP5.4.1 – Tilted Nested Rectangular Grid for Fresno Air Terminal Point – Small

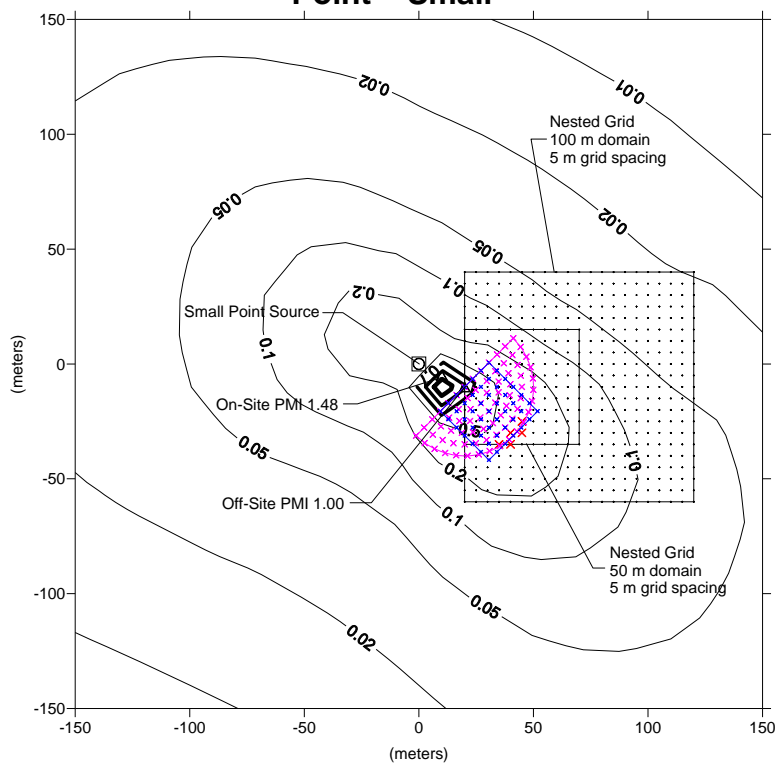


Table AP5.4.1 – Spatial Average – Fresno Air Terminal – Small Point Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.92	Polar, R = 5m
100	0.70	0.83	-	Rectangular, 10m x 10m
157	-	-	0.79	Polar, R = 10m
353	-	-	0.67	Polar, R = 15m
400	0.56	0.67	-	Rectangular, 20m x 20m
628	-	-	0.58	Polar, R = 20m
900	0.44	0.54	-	Rectangular, 30m x 30m
982	-	-	0.50	Polar, R = 25m

Figure AP5.4.2 – Tilted Nested Rectangular Grid for Fresno Air Terminal Volume – Small

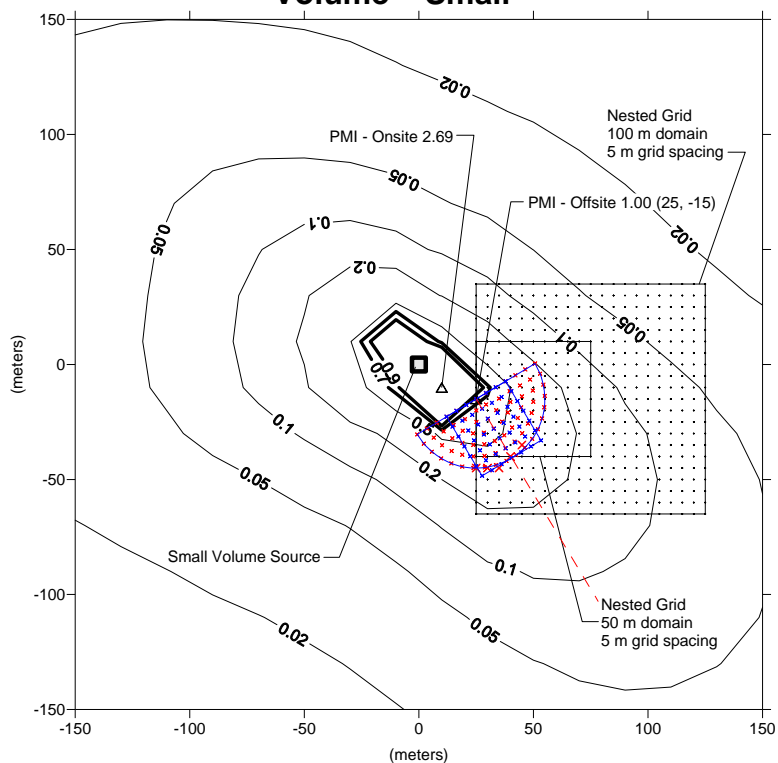


Table AP54.2 – Spatial Average – Fresno Air Terminal – Small Volume Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.93	Polar, R = 5m
100	0.76	0.82	-	Rectangular, 10m x 10m
157	-	-	0.83	Polar, R = 10m
353	-	-	0.73	Polar, R = 15m
400	0.60	0.67	-	Rectangular, 20m x 20m
628	-	-	0.63	Polar, R = 20m
900	0.47	0.55	-	Rectangular, 30m x 30m
982	-	-	0.55	Polar, R = 25m

Figure AP5.4.3 – Tilted Nested Rectangular Grid for Fresno Air Terminal Area – Small

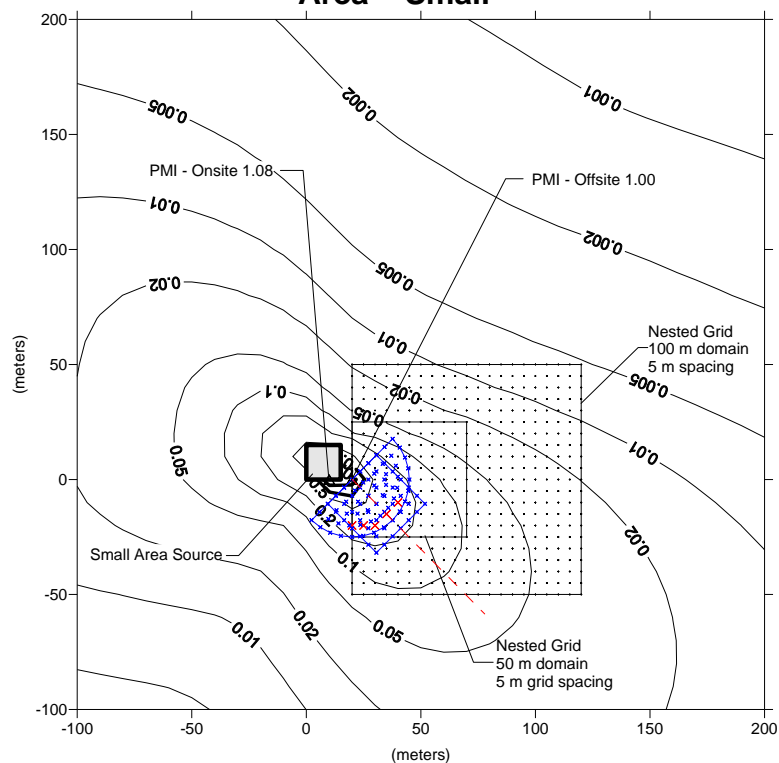


Table AP5.4.3 – Spatial Average – Fresno Air Terminal – Small Area Source

Nested Grid Domain in m ²	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.83	Polar, R = 5m
100	0.65	0.69	-	Rectangular, 10m x 10m
157	-	-	0.65	Polar, R = 10m
353	-	-	0.51	Polar, R = 15m
400	0.44	0.49	-	Rectangular, 20m x 20m
628	-	-	0.41	Polar, R = 20m
900	0.32	0.37	-	Rectangular, 30m x 30m
982	-	-	0.34	Polar, R = 25m

Appendix D

Food Codes for NHANES

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
25	Spinach souffle	72125240
25	Broccoli casserole (broccoli, noodles, and cream sauce)	72202010
25	Broccoli casserole (broccoli, rice, cheese, and mushroom sau	72202020
25	Broccoli, batter-dipped and fried	72202030
25	Broccoli soup	72302000
25	Broccoli cheese soup, prepared with milk	72302100
25	Spinach soup	72307000
25	Dark-green leafy vegetable soup with meat, Oriental style	72308000
25	Dark-green leafy vegetable soup, meatless, Oriental style	72308500
25	Raw vegetable, NFS	75100250
25	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25	Vegetable combination (including carrots, broccoli, and/or d	75440100
25	Vegetable tempura	75440200
25	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25	Vegetable combinations (including carrots, broccoli, and/or	75440500
25	Vegetable combination (including carrots, broccoli, and/or d	75450500
25	Vegetable combinations (including carrots, broccoli, and/or	75460800
25	Vegetable soup, home recipe	75649110
25	Vegetable noodle soup, home recipe	75649150
25	Vegetable beef soup, home recipe	75652010
25	Vegetable beef soup with noodles or pasta, home recipe	75652040
25	Vegetable beef soup with rice, home recipe	75652050
33	Seven-layer salad (lettuce salad made with a combination of	75145000
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
50	Cabbage soup	75601200
50	Cabbage with meat soup	75601210
50	Broccoli and chicken, baby food, strained	76604000
75	Spinach, cooked, NS as to form, with cheese sauce	72125250
75	Turnip greens with roots, cooked, NS as to form, fat not add	72128410

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
75	Broccoli, cooked, NS as to form, with cheese sauce	72201230
75	Broccoli, cooked, from fresh, with cheese sauce	72201231
75	Broccoli, cooked, from frozen, with cheese sauce	72201232
75	Broccoli, cooked, NS as to form, with cream sauce	72201250
75	Broccoli, cooked, from fresh, with cream sauce	72201251
75	Cabbage salad or coleslaw with apples and/or raisins, with dressing	75141100
75	Cabbage salad or coleslaw with pineapple, with dressing	75141200
75	Lettuce, salad with assorted vegetables including tomatoes and	75143000
75	Lettuce, salad with cheese, tomato and/or carrots, with or without	75143200
75	Lettuce salad with egg, cheese, tomato, and/or carrots, with	75143350
75	Spinach, creamed, baby food, strained	76102010
100	Beet greens, cooked, fat not added in cooking	72101210
100	Chard, cooked, fat not added in cooking	72104210
100	Chard, cooked, fat added in cooking	72104220
100	Collards, raw	72107100
100	Collards, cooked, NS as to form, NS as to fat added in cooking	72107200
100	Collards, cooked, from fresh, NS as to fat added in cooking	72107201
100	Collards, cooked, from fresh, fat not added in cooking	72107211
100	Collards, cooked, NS as to form, fat added in cooking	72107220
100	Collards, cooked, from fresh, fat added in cooking	72107221
100	Collards, cooked, from frozen, fat added in cooking	72107222
100	Greens, cooked, from fresh, fat not added in cooking	72118211
100	Greens, cooked, NS as to form, fat added in cooking	72118220
100	Greens, cooked, from fresh, fat added in cooking	72118221
100	Kale, cooked, NS as to form, NS as to fat added in cooking	72119200
100	Kale, cooked, from fresh, fat not added in cooking	72119211
100	Kale, cooked, NS as to form, fat added in cooking	72119220
100	Kale, cooked, from fresh, fat added in cooking	72119221
100	Mustard greens, cooked, NS as to form, NS as to fat added in cooking	72122200
100	Mustard greens, cooked, from fresh, NS as to fat added in cooking	72122201
100	Mustard greens, cooked, from fresh, fat not added in cooking	72122211
100	Mustard greens, cooked, from canned, fat not added in cooking	72122213
100	Mustard greens, cooked, from fresh, fat added in cooking	72122221

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Mustard greens, cooked, from frozen, fat added in cooking	72122222
100	Mustard greens, cooked, from canned, fat added in cooking	72122223
100	Poke greens, cooked, fat not added in cooking	72123010
100	Poke greens, cooked, fat added in cooking	72123020
100	Radicchio, raw	72124100
100	Spinach, raw	72125100
100	Spinach, cooked, NS as to form, NS as to fat added in cookin	72125200
100	Spinach, cooked, from fresh, NS as to fat added in cooking	72125201
100	Spinach, cooked, from frozen, NS as to fat added in cooking	72125202
100	Spinach, cooked, NS as to form, fat not added in cooking	72125210
100	Spinach, cooked, from fresh, fat not added in cooking	72125211
100	Spinach, cooked, from frozen, fat not added in cooking	72125212
100	Spinach, cooked, NS as to form, fat added in cooking	72125220
100	Spinach, cooked, from fresh, fat added in cooking	72125221
100	Spinach, cooked, from frozen, fat added in cooking	72125222
100	Spinach, NS as to form, creamed	72125230
100	Turnip greens, cooked, from fresh, fat not added in cooking	72128211
100	Turnip greens, cooked, NS as to form, fat added in cooking	72128220
100	Turnip greens, cooked, from fresh, fat added in cooking	72128221
100	Turnip greens, cooked, from frozen, fat added in cooking	72128222
100	Watercress, raw	72130100
100	Broccoli, raw	72201100
100	Broccoli, cooked, NS as to form, NS as to fat added in cooki	72201200
100	Broccoli, cooked, from fresh, NS as to fat added in cooking	72201201
100	Broccoli, cooked, from frozen, NS as to fat added in cooking	72201202
100	Broccoli, cooked, NS as to form, fat not added in cooking	72201210
100	Broccoli, cooked, from fresh, fat not added in cooking	72201211
100	Broccoli, cooked, from frozen, fat not added in cooking	72201212
100	Broccoli, cooked, NS as to form, fat added in cooking	72201220
100	Broccoli, cooked, from fresh, fat added in cooking	72201221
100	Broccoli, cooked, from frozen, fat added in cooking	72201222
100	Sprouts, NFS	75100300
100	Alfalfa sprouts, raw	75100500

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Artichoke, Jerusalem, raw	75100750
100	Cabbage, green, raw	75103000
100	Cabbage, Chinese, raw	75104000
100	Cabbage, red, raw	75105000
100	Cauliflower, raw	75107000
100	Celery, raw	75109000
100	Chives, raw	75109500
100	Cilantro, raw	75109550
100	Lettuce, raw	75113000
100	Lettuce, Boston, raw	75113060
100	Lettuce, arugula, raw	75113080
100	Mixed salad greens, raw	75114000
100	Parsley, raw	75119000
100	Broccoli salad with cauliflower, cheese, bacon bits, and dre	75140500
100	Cabbage salad or coleslaw, with dressing	75141000
100	Artichoke, globe (French), cooked, NS as to form, NS as to f	75201000
100	Artichoke, globe (French), cooked, NS as to form, fat not ad	75201010
100	Artichoke, globe (French), cooked, from fresh, fat not added	75201011
100	Artichoke, globe (French), cooked, from canned, fat not adde	75201013
100	Artichoke, globe (French), cooked, NS as to form, fat added	75201020
100	Artichoke, globe (French), cooked, from fresh, fat added in	75201021
100	Artichoke salad in oil	75201030
100	Brussels sprouts, cooked, NS as to form, fat not added in co	75209010
100	Brussels sprouts, cooked, from fresh, fat not added in cooki	75209011
100	Brussels sprouts, cooked, from frozen, fat not added in cook	75209012
100	Brussels sprouts, cooked, from fresh, fat added in cooking	75209021
100	Brussels sprouts, cooked, from frozen, fat added in cooking	75209022
100	Cabbage, Chinese, cooked, NS as to fat added in cooking	75210000
100	Cabbage, Chinese, cooked, fat not added in cooking	75210010
100	Cabbage, Chinese, cooked, fat added in cooking	75210020
100	Cabbage, green, cooked, NS as to fat added in cooking	75211010
100	Cabbage, green, cooked, fat not added in cooking	75211020
100	Cabbage, green, cooked, fat added in cooking	75211030

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Cabbage, red, cooked, fat not added in cooking	75212010
100	Cauliflower, cooked, NS as to form, NS as to fat added in co	75214000
100	Cauliflower, cooked, from fresh, NS as to fat added in cooki	75214001
100	Cauliflower, cooked, from frozen, NS as to fat added in cook	75214002
100	Cauliflower, cooked, NS as to form, fat not added in cooking	75214010
100	Cauliflower, cooked, from fresh, fat not added in cooking	75214011
100	Cauliflower, cooked, from frozen, fat not added in cooking	75214012
100	Cauliflower, cooked, NS as to form, fat added in cooking	75214020
100	Cauliflower, cooked, from fresh, fat added in cooking	75214021
100	Cauliflower, cooked, from frozen, fat added in cooking	75214022
100	Lettuce, cooked, fat not added in cooking	75220050
100	Parsley, cooked (assume fat not added in cooking)	75221210
100	Cauliflower, batter-dipped, fried	75409020
100	Cabbage, red, pickled	75502510
100	Cabbage, Kim Chee style	75502520

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetable beef soup, home recipe	75652010
12.5	Vegetable beef soup with noodles or pasta, home recipe	75652040
12.5	Vegetable beef soup with rice, home recipe	75652050
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Raw vegetable, NFS	75100250
25.0	Cabbage salad or coleslaw with apples and/or raisins, with d	75141100
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable tempura	75440200
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010
33.0	Grape juice	64116020
33.0	Peach juice, with sugar	64122030
33.0	Apple-banana juice, baby food	67203200
33.0	Apple-cranberry juice, baby food	67203450
33.0	Tomato soup, NFS	74601000
33.0	Tomato soup, prepared with water	74602010

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mushroom soup, NFS	75607000
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Bananas with apples and pears, baby food, strained	67106010
50.0	Pears and pineapple, baby food, strained	67114010
50.0	Pears and pineapple, baby food, junior	67114020
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500
50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Chiles rellenos, cheese-filled (stuffed chili peppers)	75410500
50.0	Chiles rellenos, filled with meat and cheese (stuffed chili	75410530
50.0	Minestrone soup, home recipe	75651000
50.0	Jelly, all flavors	91401000
50.0	Jam, preserves, all flavors	91402000
50.0	Jelly, dietetic, all flavors, sweetened with artificial swee	91405000
50.0	Jelly, reduced sugar, all flavors	91405500
66.0	Fruit juice, NFS	64100100
66.0	Apple cider	64101010
66.0	Apple juice	64104010
66.0	Prune juice	64132010

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
66.0	Prune juice, unsweetened	64132020
66.0	Strawberry juice	64132500
66.0	Apple juice, baby food	67202000
66.0	Apple with other fruit juice, baby food	67203000
66.0	Apple-cherry juice, baby food	67203400
66.0	Apple-grape juice, baby food	67203500
66.0	Apple-prune juice, baby food	67203700
66.0	Grape juice, baby food	67203800
66.0	Mixed fruit juice, not citrus, baby food	67204000
66.0	Pear juice, baby food	67212000
66.0	Tomato juice	74301100
66.0	Tomato and vegetable juice, mostly tomato	74303000
66.0	Mixed vegetable juice (vegetables other than tomato)	75132000
66.0	Celery juice	75132100
66.0	Gazpacho	75604600
100.0	Fruit, dried, NFS (assume uncooked)	62101000
100.0	Fruit mixture, dried (mixture includes three or more of the	62101050
100.0	Apple, dried, uncooked	62101100
100.0	Apple, dried, cooked, NS as to sweetened or unsweetened; swe	62101200
100.0	Apricot, dried, uncooked	62104100
100.0	Pear, dried, cooked, with sugar	62119230
100.0	Prune, dried, uncooked	62122100
100.0	Prune, dried, cooked, NS as to sweetened or unsweetened; swe	62122200
100.0	Prune, dried, cooked, unsweetened	62122220
100.0	Prune, dried, cooked, with sugar	62122230
100.0	Raisins	62125100
100.0	Raisins, cooked	62125110
100.0	Apple, raw	63101000
100.0	Applesauce, stewed apples, NS as to sweetened or unsweetened	63101110
100.0	Applesauce, stewed apples, unsweetened	63101120
100.0	Applesauce, stewed apples, with sugar	63101130
100.0	Applesauce, stewed apples, sweetened with low calorie sweete	63101140

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Applesauce with other fruits	63101150
100.0	Apple, cooked or canned, with syrup	63101210
100.0	Apple, baked, NS as to added sweetener	63101310
100.0	Apple, baked, unsweetened	63101320
100.0	Apple, baked, with sugar	63101330
100.0	Apple, pickled	63101420
100.0	Apple, fried	63101500
100.0	Apricot, raw	63103010
100.0	Apricot, cooked or canned, NS as to sweetened or unsweetened	63103110
100.0	Apricot, cooked or canned, in light syrup	63103140
100.0	Apricot, cooked or canned, drained solids	63103150
100.0	Apricot, cooked or canned, juice pack	63103170
100.0	Cherry pie filling	63113030
100.0	Cherries, sweet, raw (Queen Anne, Bing)	63115010
100.0	Cherries, sweet, cooked or canned, drained solids	63115150
100.0	Fig, raw	63119010
100.0	Grapes, raw, NS as to type	63123000
100.0	Grapes, European type, adherent skin, raw	63123010
100.0	Grapes, seedless, cooked or canned, unsweetened, water pack	63123120
100.0	Mango, raw	63129010
100.0	Mango, cooked	63129030
100.0	Nectarine, raw	63131010
100.0	Nectarine, cooked	63131110
100.0	Peach, raw	63135010
100.0	Peach, cooked or canned, NS as to sweetened or unsweetened;	63135110
100.0	Peach, cooked or canned, in heavy syrup	63135130
100.0	Peach, cooked or canned, in light or medium syrup	63135140
100.0	Peach, cooked or canned, drained solids	63135150
100.0	Peach, cooked or canned, juice pack	63135170
100.0	Peach, frozen, NS as to added sweetener	63135610
100.0	Peach, frozen, unsweetened	63135620
100.0	Peach, frozen, with sugar	63135630

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Pear, raw	63137010
100.0	Pear, Japanese, raw	63137050
100.0	Pear, cooked or canned, NS as to sweetened or unsweetened; s	63137110
100.0	Pear, cooked or canned, in heavy syrup	63137130
100.0	Pear, cooked or canned, in light syrup	63137140
100.0	Pear, cooked or canned, drained solids	63137150
100.0	Pear, cooked or canned, juice pack	63137170
100.0	Persimmon, raw	63139010
100.0	Plum, raw	63143010
100.0	Plum, cooked or canned, in light syrup	63143140
100.0	Plum, pickled	63143650
100.0	Rhubarb, frozen, with sugar	63147620
100.0	SUGAR APPLE, SWEETSOP (ANON), RAW	63148010
100.0	Blackberries, raw	63201010
100.0	Blackberries, cooked or canned, NS as to sweetened or unswee	63201110
100.0	Raspberries, raw, NS as to color	63219000
100.0	Raspberries, red, raw	63219020
100.0	Raspberries, cooked or canned, NS as to sweetened or unsweet	63219110
100.0	Raspberries, frozen, unsweetened	63219610
100.0	Strawberries, raw	63223020
100.0	Strawberries, raw, with sugar	63223030
100.0	Strawberries, cooked or canned, NS as to sweetened or unswee	63223110
100.0	Strawberries, cooked or canned, unsweetened, water pack	63223120
100.0	Strawberries, cooked or canned, in syrup	63223130
100.0	Strawberries, frozen, NS as to added sweetener	63223600
100.0	Strawberries, frozen, unsweetened	63223610
100.0	Strawberries, frozen, with sugar	63223620
100.0	Fruit cocktail or mix (excluding citrus fruits), raw	63311000
100.0	Apple salad with dressing	63401010
100.0	Apple, candied	63401060
100.0	Fruit salad (excluding citrus fruits) with salad dressing or	63402950
100.0	Fruit salad (excluding citrus fruits) with cream	63402960

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Fruit salad (excluding citrus fruits) with cream substitute	63402970
100.0	Fruit salad (excluding citrus fruits) with marshmallows	63402980
100.0	Fruit salad (excluding citrus fruits) with pudding	63403000
100.0	Fruit salad (including citrus fruits) with salad dressing or	63403010
100.0	Fruit salad (including citrus fruit) with cream	63403020
100.0	Fruit salad (including citrus fruits) with marshmallows	63403040
100.0	Chutney	63409020
100.0	Tomato and okra, cooked, NS as to fat added in cooking	74504000
100.0	Tomato and okra, cooked, fat not added in cooking	74504010
100.0	Tomato and okra, cooked, fat added in cooking	74504020
100.0	Tomato and celery, cooked, fat not added in cooking	74504150
100.0	Cucumber salad with creamy dressing	75142500
100.0	Cucumber salad made with cucumber, oil, and vinegar	75142550
100.0	Cucumber salad made with cucumber and vinegar	75142600
100.0	Cucumber pickles, dill	75503010
100.0	Cucumber pickles, relish	75503020
100.0	Cucumber pickles, sour	75503030
100.0	Cucumber pickles, sweet	75503040
100.0	Cucumber pickles, fresh	75503050
100.0	Mustard pickles	75503100
100.0	Cucumber pickles, dill, reduced salt	75503110

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Carrots and beef, baby food, strained	76602000
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Lemon pie filling	61113500
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable sticks, breaded (including corn, carrots, and gree	75440170
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Fruit sauce	91361020
33.0	Strawberry-banana-orange juice	61226000

% Protected Produce in Food Item	Food Item Description	USDA Food Code
33.0	Vegetable stew without meat	75439010
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Orange and banana juice	61219000
50.0	Pineapple-orange juice, NFS	61225000
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and onions, cooked, fat added in cooking	75315120
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Pinacbet (eggplant with tomatoes, bitter melon, etc.)	75340300
50.0	Eggplant, batter-dipped, fried	75412010
50.0	Eggplant dip	75412030
50.0	Eggplant parmesan casserole, regular	75412060
50.0	Pea salad	75416500
50.0	Pea salad with cheese	75416600
50.0	Squash, summer, yellow or green, breaded or battered, baked	75418000
50.0	Squash, summer, yellow or green, breaded or battered, fried	75418010
50.0	Pea soup, NFS	75609000
50.0	Carrots and peas, baby food, strained	76202000
100.0	Almonds, NFS	42100100
100.0	Almonds, unroasted	42101000
100.0	Chestnuts, roasted	42105000
100.0	Filberts, hazelnuts	42107000
100.0	Pecans	42112000
100.0	Walnuts	42116000

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Pumpkin and/or squash seeds, hulled, roasted, salted	43101100
100.0	Grapefruit, raw	61101010
100.0	Grapefruit, canned or frozen, NS as to sweetened or unsweete	61101200
100.0	Grapefruit, canned or frozen, in light syrup	61101230
100.0	Lemon, raw	61113010
100.0	Lime, raw	61116010
100.0	Orange, raw	61119010
100.0	Orange, mandarin, canned or frozen, NS as to sweetened or un	61122300
100.0	Orange, mandarin, canned or frozen, juice pack	61122320
100.0	Orange, mandarin, canned or frozen, in light syrup	61122330
100.0	Orange, mandarin, canned or frozen, drained	61122350
100.0	Tangerine, raw	61125010
100.0	Grapefruit juice, freshly squeezed	61201010
100.0	Lemon juice, NS as to form	61204000
100.0	Lemon juice, fresh	61204010
100.0	Lemon juice, frozen	61204600
100.0	Lime juice, NS as to form	61207000
100.0	Lime juice, fresh	61207010
100.0	Lime juice, frozen	61207600
100.0	Orange juice, NFS	61210000
100.0	Orange juice, freshly squeezed	61210010
100.0	Tangerine juice, NFS	61213000
100.0	Avocado, raw	63105010
100.0	Cantaloupe (muskmelon), raw	63109010
100.0	Cantaloupe, frozen (balls)	63109610
100.0	Kiwi fruit, raw	63126500
100.0	Honeydew melon, raw	63127010
100.0	Honeydew, frozen (balls)	63127610
100.0	Papaya, raw	63133010
100.0	Papaya, cooked or canned, in sugar or syrup	63133100
100.0	Pomegranate, raw	63145010
100.0	Watermelon, raw	63149010

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Guacamole with tomatoes	63408010
100.0	Guacamole with tomatoes and chili peppers	63408200
100.0	Guacamole, NFS	63409010
100.0	Pumpkin, cooked, from fresh, fat not added in cooking	73201011
100.0	Pumpkin, cooked, from canned, fat not added in cooking	73201013
100.0	Pumpkin, cooked, NS as to form, fat added in cooking	73201020
100.0	Pumpkin, cooked, from fresh, fat added in cooking	73201021
100.0	Calabaza (Spanish pumpkin), cooked	73210010
100.0	Squash, winter type, mashed, NS as to fat or sugar added in	73301000
100.0	Squash, winter type, mashed, no fat or sugar added in cookin	73301010
100.0	Squash, winter type, mashed, fat added in cooking, no sugar	73301020
100.0	Squash, winter type, baked, NS as to fat or sugar added in c	73303000
100.0	Squash, winter type, baked, no fat or sugar added in cooking	73303010
100.0	Squash, winter type, baked, fat added in cooking, no sugar a	73303020
100.0	Squash, winter, baked with cheese	73305010
100.0	Peas, green, raw	75120000
100.0	Squash, summer, yellow, raw	75128000
100.0	Squash, summer, green, raw	75128010
100.0	Beans, lima, immature, cooked, NS as to form, NS as to fat a	75204000
100.0	Beans, lima, immature, cooked, from fresh, fat not added in	75204011
100.0	Beans, lima, immature, cooked, from frozen, fat not added in	75204012
100.0	Beans, lima, immature, cooked, NS as to form, fat added in c	75204020
100.0	Beans, lima, immature, cooked, from fresh, fat added in cook	75204021
100.0	Beans, lima, immature, cooked, from frozen, fat added in coo	75204022
100.0	Bitter melon, cooked, fat added in cooking	75208310
100.0	Cactus, cooked, NS as to fat added in cooking	75213100
100.0	Cactus, cooked, fat not added in cooking	75213110
100.0	Cactus, cooked, fat added in cooking	75213120
100.0	Christophine, cooked, fat not added in cooking	75215510
100.0	Corn, cooked, NS as to form, NS as to color, NS as to fat ad	75216000
100.0	Corn, cooked, from fresh, NS as to color, NS as to fat added	75216001
100.0	Corn, cooked, from frozen, NS as to color, NS as to fat adde	75216002
100.0	Corn, cooked, NS as to form, NS as to color, fat not added i	75216010

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, cooked, from fresh, NS as to color, fat not added in c	75216011
100.0	Corn, cooked, from frozen, NS as to color, fat not added in	75216012
100.0	Corn, cooked, NS as to form, NS as to color, fat added in co	75216020
100.0	Corn, cooked, from fresh, NS as to color, fat added in cooki	75216021
100.0	Corn, cooked, from frozen, NS as to color, fat added in cook	75216022
100.0	Corn, NS as to form, NS as to color, cream style	75216050
100.0	Corn, yellow, cooked, NS as to form, NS as to fat added in c	75216100
100.0	Corn, yellow, cooked, from fresh, NS as to fat added in cook	75216101
100.0	Corn, yellow, cooked, from frozen, NS as to fat added in coo	75216102
100.0	Corn, yellow, cooked, NS as to form, fat not added in cookin	75216110
100.0	Corn, yellow, cooked, from fresh, fat not added in cooking	75216111
100.0	Corn, yellow, cooked, from frozen, fat not added in cooking	75216112
100.0	Corn, yellow, cooked, NS as to form, fat added in cooking	75216120
100.0	Corn, yellow, cooked, from fresh, fat added in cooking	75216121
100.0	Corn, yellow, cooked, from frozen, fat added in cooking	75216122
100.0	Corn, yellow, NS as to form, cream style	75216150
100.0	Corn, yellow and white, cooked, NS as to form, NS as to fat	75216160
100.0	Corn, yellow and white, cooked, from fresh, NS as to fat add	75216161
100.0	Corn, yellow and white, cooked, NS as to form, fat not added	75216170
100.0	Corn, yellow and white, cooked, from fresh, fat not added in	75216171
100.0	Corn, yellow and white, cooked, from fresh, fat added in coo	75216181
100.0	Corn, white, cooked, NS as to form, NS as to fat added in co	75216200
100.0	Corn, white, cooked, from fresh, NS as to fat added in cooki	75216201
100.0	Corn, white, cooked, NS as to form, fat not added in cooking	75216210
100.0	Corn, white, cooked, from fresh, fat not added in cooking	75216211
100.0	Corn, white, cooked, from frozen, fat not added in cooking	75216212
100.0	Corn, white, cooked, from fresh, fat added in cooking	75216221
100.0	Corn, white, cooked, from frozen, fat added in cooking	75216222
100.0	Hominy, cooked, fat not added in cooking	75217500
100.0	Hominy, cooked, fat added in cooking	75217520
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223000
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223020

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223021
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223022
100.0	Peas, green, cooked, NS as to form, NS as to fat added in co	75224010
100.0	Peas, green, cooked, from fresh, NS as to fat added in cooki	75224011
100.0	Peas, green, cooked, from frozen, NS as to fat added in cook	75224012
100.0	Peas, green, cooked, NS as to form, fat not added in cooking	75224020
100.0	Peas, green, cooked, from fresh, fat not added in cooking	75224021
100.0	Peas, green, cooked, from frozen, fat not added in cooking	75224022
100.0	Peas, green, cooked, NS as to form, fat added in cooking	75224030
100.0	Peas, green, cooked, from fresh, fat added in cooking	75224031
100.0	Peas, green, cooked, from frozen, fat added in cooking	75224032
100.0	Pigeon peas, cooked, NS as to form, fat not added in cooking	75225010
100.0	Squash, summer, cooked, NS as to form, NS as to fat added in	75233000
100.0	Squash, summer, cooked, from fresh, NS as to fat added in co	75233001
100.0	Squash, summer, cooked, from frozen, NS as to fat added in c	75233002
100.0	Squash, summer, cooked, NS as to form, fat not added in cook	75233010
100.0	Squash, summer, cooked, from fresh, fat not added in cooking	75233011
100.0	Squash, summer, cooked, from frozen, fat not added in cookin	75233012
100.0	Squash, summer, cooked, NS as to form, fat added in cooking	75233020
100.0	Squash, summer, cooked, from fresh, fat added in cooking	75233021
100.0	Beans, lima and corn (succotash), cooked, fat not added in c	75301110
100.0	Beans, lima and corn (succotash), cooked, fat added in cooki	75301120
100.0	Peas and corn, cooked, NS as to fat added in cooking	75315000
100.0	Peas and corn, cooked, fat not added in cooking	75315010
100.0	Peas and corn, cooked, fat added in cooking	75315020
100.0	Squash, baby food, strained	76205010
100.0	Corn, creamed, baby food, strained	76405010
100.0	Corn, creamed, baby food, junior	76405020
100.0	Peas, baby food, NS as to strained or junior	76409000
100.0	Peas, baby food, strained	76409010
100.0	Peas, baby food, junior	76409020
100.0	Marmalade, all flavors	91404000

% Protected Produce in Food Item	Food Item Description	USDA Food Code
12.5	Beet soup (borscht)	75601100
12.5	Leek soup, cream of, prepared with milk	75605010
12.5	Onion soup, French	75608100
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
12.5	Puerto Rican stew (Sancocho)	77563010
25.0	Raw vegetable, NFS	75100250
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010

% Protected Produce in Food Item	Food Item Description	USDA Food Code
33.0	Mixed vegetable juice (vegetables other than tomato)	75132000
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000

Table D.4 Food Codes for Root Vegetables

% Root Produce in Food Item	Food Item Description	USDA Food Code
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
50.0	Potato pancake	71701000
50.0	Norwegian Lefse, potato and flour pancake	71701500
50.0	Stewed potatoes, Mexican style (Papas guisadas)	71703000
50.0	Stewed potatoes with tomatoes, Mexican style (Papas guisadas)	71703040
50.0	Stewed potatoes with tomatoes	71704000
50.0	Potato soup, NS as to made with milk or water	71801000
50.0	Potato soup, cream of, prepared with milk	71801010
50.0	Potato soup, prepared with water	71801020
50.0	Potato soup, instant, made from dry mix	71801040
50.0	Potato and cheese soup	71801100
50.0	Macaroni and potato soup	71802010
50.0	Potato chowder	71803010
50.0	Peas and carrots, cooked, NS as to form, NS as to fat added	73111200
50.0	Peas and carrots, cooked, from fresh, NS as to fat added in	73111201
50.0	Peas and carrots, cooked, from frozen, NS as to fat added in	73111202
50.0	Peas and carrots, cooked, NS as to form, fat not added in co	73111210
50.0	Peas and carrots, cooked, from fresh, fat not added in cooki	73111211
50.0	Peas and carrots, cooked, from frozen, fat not added in cook	73111212
50.0	Peas and carrots, cooked, NS as to form, fat added in cookin	73111220
50.0	Peas and carrots, cooked, from fresh, fat added in cooking	73111221
50.0	Peas and carrots, cooked, from frozen, fat added in cooking	73111222
50.0	Carrot soup, cream of, prepared with milk	73501000
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500
50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510

50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Onion rings, NS as to form, batter-dipped, baked or fried	75415020
50.0	Onion rings, from fresh, batter-dipped, baked or fried	75415021
50.0	Carrots and peas, baby food, strained	76202000
50.0	Carrots and beef, baby food, strained	76602000
50.0	Sweetpotatoes and chicken, baby food, strained	76604500
75.0	White potato, cooked, with cheese	71301020
75.0	White potato, cooked, with ham and cheese	71301120
75.0	White potato, scalloped	71305010
75.0	White potato, scalloped, with ham	71305110
75.0	Carrots, cooked, from fresh, creamed	73102231
75.0	Carrots, cooked, NS as to form, glazed	73102240
75.0	Carrots, cooked, from fresh, glazed	73102241
75.0	Carrots, cooked, from frozen, glazed	73102242
75.0	Carrots, cooked, from fresh, with cheese sauce	73102251
75.0	Carrots in tomato sauce	73111400
100.0	White potato, NFS	71000100
100.0	White potato, baked, peel not eaten	71101000
100.0	White potato, baked, peel eaten, NS as to fat added in cooki	71101100
100.0	White potato, baked, peel eaten, fat not added in cooking	71101110
100.0	White potato, baked, peel eaten, fat added in cooking	71101120
100.0	White potato skins, with adhering flesh, baked	71101150
100.0	White potato, boiled, without peel, NS as to fat added in co	71103000
100.0	White potato, boiled, without peel, fat not added in cooking	71103010
100.0	White potato, boiled, without peel, fat added in cooking	71103020
100.0	White potato, boiled, with peel, NS as to fat added in cooki	71103100
100.0	White potato, boiled, with peel, fat not added in cooking	71103110
100.0	White potato, boiled, with peel, fat added in cooking	71103120
100.0	White potato, boiled, without peel, canned, low sodium, fat	71103210
100.0	White potato, roasted, NS as to fat added in cooking	71104000
100.0	White potato, roasted, fat not added in cooking	71104010
100.0	White potato, roasted, fat added in cooking	71104020
100.0	White potato, sticks	71205000

100.0	White potato skins, chips	71211000
100.0	White potato, french fries, NS as to from fresh or frozen	71401000
100.0	White potato, french fries, from fresh, deep fried	71401010
100.0	White potato, french fries, from frozen, oven baked	71401020
100.0	White potato, french fries, from frozen, deep fried	71401030
100.0	White potato, french fries, breaded or battered	71402040
100.0	White potato, home fries	71403000
100.0	White potato, home fries, with green or red peppers and onion	71403500
100.0	White potato, hash brown, NS as to from fresh, frozen, or dr	71405000
100.0	White potato, hash brown, from fresh	71405010
100.0	White potato, hash brown, from frozen	71405020
100.0	White potato, hash brown, with cheese	71405100
100.0	White potato skins, with adhering flesh, fried	71410000
100.0	White potato skins, with adhering flesh, fried, with cheese	71410500
100.0	White potato skins, with adhering flesh, fried, with cheese	71411000
100.0	White potato, mashed, NFS	71501000
100.0	White potato, from fresh, mashed, made with milk	71501010
100.0	White potato, from fresh, mashed, made with milk, sour cream	71501015
100.0	White potato, from fresh, mashed, made with milk and fat	71501020
100.0	White potato, from fresh, mashed, made with fat	71501030
100.0	White potato, from fresh, mashed, made with milk, fat and ch	71501050
100.0	White potato, from fresh, mashed, not made with milk or fat	71501080
100.0	White potato, from fresh, mashed, NS as to milk or fat	71501310
100.0	White potato, patty	71503010
100.0	White potato, puffs	71505000
100.0	White potato, stuffed, baked, peel not eaten, NS as to top	71507000
100.0	White potato, stuffed, baked, peel not eaten, stuffed with s	71507010
100.0	White potato, stuffed, baked, peel not eaten, stuffed with c	71507020
100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71507040
100.0	White potato, stuffed, baked, peel eaten, stuffed with sour	71508010
100.0	White potato, stuffed, baked, peel eaten, stuffed with chees	71508020
100.0	White potato, stuffed, baked, peel eaten, stuffed with chili	71508030
100.0	White potato, stuffed, baked, peel eaten, stuffed with brocc	71508040
100.0	White potato, stuffed, baked, peel eaten, stuffed with meat	71508050
100.0	White potato, stuffed, baked, peel eaten, stuffed with bacon	71508060

100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71508070
100.0	Potato salad with egg	71601010
100.0	Potato salad, German style	71602010
100.0	Potato salad	71603010
100.0	Carrots, raw	73101010
100.0	Carrots, raw, salad	73101110
100.0	Carrots, raw, salad with apples	73101210
100.0	Carrots, cooked, NS as to form, NS as to fat added in cookin	73102200
100.0	Carrots, cooked, from fresh, NS as to fat added in cooking	73102201
100.0	Carrots, cooked, from frozen, NS as to fat added in cooking	73102202
100.0	Carrots, cooked, NS as to form, fat not added in cooking	73102210
100.0	Carrots, cooked, from fresh, fat not added in cooking	73102211
100.0	Carrots, cooked, from frozen, fat not added in cooking	73102212
100.0	Carrots, cooked, NS as to form, fat added in cooking	73102220
100.0	Carrots, cooked, from fresh, fat added in cooking	73102221
100.0	Carrots, cooked, from frozen, fat added in cooking	73102222
100.0	Sweetpotato, NFS	73401000
100.0	Sweetpotato, baked, peel eaten, fat not added in cooking	73402010
100.0	Sweetpotato, baked, peel eaten, fat added in cooking	73402020
100.0	Sweetpotato, baked, peel not eaten, NS as to fat added in co	73403000
100.0	Sweetpotato, baked, peel not eaten, fat not added in cooking	73403010
100.0	Sweetpotato, baked, peel not eaten, fat added in cooking	73403020
100.0	Sweetpotato, boiled, without peel, NS as to fat added in coo	73405000
100.0	Sweetpotato, boiled, without peel, fat not added in cooking	73405010
100.0	Sweetpotato, boiled, without peel, fat added in cooking	73405020
100.0	Sweetpotato, boiled, with peel, fat not added in cooking	73405110
100.0	Sweetpotato, boiled, with peel, fat added in cooking	73405120
100.0	Sweetpotato, candied	73406000
100.0	Sweetpotato, canned, NS as to syrup	73407000
100.0	Sweetpotato, canned without syrup	73407010
100.0	Sweetpotato, canned in syrup, with fat added in cooking	73407030
100.0	Sweetpotato, casserole or mashed	73409000
100.0	Sweetpotato, fried	73410110
100.0	Beets, raw	75102500
100.0	Garlic, raw	75111500
100.0	Jicama, raw	75111800

100.0	Onions, young green, raw	75117010
100.0	Onions, mature, raw	75117020
100.0	Radish, raw	75125000
100.0	Turnip, raw	75129000
100.0	Beets, cooked, NS as to form, NS as to fat added in cooking	75208000
100.0	Beets, cooked, NS as to form, fat not added in cooking	75208010
100.0	Beets, cooked, from fresh, fat not added in cooking	75208011
100.0	Beets, cooked, NS as to form, fat added in cooking	75208020
100.0	Beets, cooked, from fresh, fat added in cooking	75208021
100.0	Garlic, cooked	75217400
100.0	Onions, mature, cooked, NS as to form, NS as to fat added in	75221000
100.0	Onions, mature, cooked, from fresh, NS as to fat added in co	75221001
100.0	Onions, mature, cooked, from frozen, NS as to fat added in c	75221002
100.0	Onions, mature, cooked, NS as to form, fat not added in cook	75221010
100.0	Onions, mature, cooked, from fresh, fat not added in cooking	75221011
100.0	Onions, mature, cooked or sauteed, NS as to form, fat added	75221020
100.0	Onions, mature, cooked or sauteed, from fresh, fat added in	75221021
100.0	Onions, mature, cooked or sauteed, from frozen, fat added in	75221022
100.0	Onions, pearl, cooked, NS as to form	75221030
100.0	Onions, pearl, cooked, from fresh	75221031
100.0	Onion, young green, cooked, NS as to form, NS as to fat adde	75221040
100.0	Onions, young green, cooked, NS as to form, fat not added in	75221050
100.0	Onions, young green, cooked, from fresh, fat not added in co	75221051
100.0	Onion, young green, cooked, from fresh, fat added in cooking	75221061
100.0	Parsnips, cooked, fat not added in cooking	75222010
100.0	Parsnips, cooked, fat added in cooking	75222020
100.0	Radish, Japanese (daikon), cooked, fat added in cooking	75227110
100.0	Turnip, cooked, from fresh, NS as to fat added in cooking	75234001
100.0	Turnip, cooked, NS as to form, fat not added in cooking	75234010
100.0	Turnip, cooked, from fresh, fat not added in cooking	75234011
100.0	Turnip, cooked, from fresh, fat added in cooking	75234021
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317000
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317010
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317020
100.0	Beets with Harvard sauce	75405010

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100.0	Beets, pickled	75500210
100.0	Carrots, baby food, NS as to strained or junior	76201000
100.0	Carrots, baby food, strained	76201010
100.0	Carrots, baby food, junior	76201020
100.0	Carrots, baby food, toddler	76201030
100.0	Sweetpotatoes, baby food, NS as to strained or junior	76209000
100.0	Sweetpotatoes, baby food, strained	76209010

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Chicken soup with noodles and potatoes, Puerto Rican style	28340220
12.5	Chicken gumbo soup	28340310
12.5	Chicken noodle soup, chunky style	28340510
12.5	Chicken soup, canned, undiluted	28340520
12.5	Chicken soup	28340530
12.5	Sweet and sour soup	28340550
12.5	Chicken soup with vegetables (broccoli, carrots, celery, pot	28340580
12.5	Chicken corn soup with noodles, home recipe	28340590
12.5	Chicken or turkey vegetable soup, stew type	28340610
12.5	Chicken vegetable soup with rice, stew type, chunky style	28340630
12.5	Chicken vegetable soup with noodles, stew type, chunky style	28340640
12.5	Chicken or turkey vegetable soup, home recipe	28340660
12.5	Chicken vegetable soup with rice, Mexican style (Sopa / Cald	28340670
12.5	Hot and sour soup	28340750
12.5	Chicken soup with vegetables and fruit, Oriental Style	28340800
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, ma	28345030
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, un	28345040
12.5	Chicken or turkey soup, cream of, NS as to prepared with mil	28345110
12.5	Chicken or turkey soup, cream of, prepared with milk	28345120
12.5	TAMALE W/ MEAT &/OR POULTRY (INCL TAMALE, NFS)	58103110
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Meat turnover, Puerto Rican style (Pastelillo de carne; Empa	58116110
12.5	Empanada, Mexican turnover, filled with meat and vegetables	58116120

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Dumpling, meat-filled	58121510
12.5	Quiche with meat, poultry or fish	58125110
12.5	Turnover, meat-filled, no gravy	58126110
12.5	Turnover, meat- and cheese-filled, no gravy	58126130
12.5	Turnover, meat- and bean-filled, no gravy	58126140
12.5	Turnover, meat- and cheese-filled, tomato-based sauce	58126150
12.5	Turnover, meat-and vegetable- filled (no potatoes, no gravy)	58126170
12.5	Dressing with chicken or turkey and vegetables	58128220
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Chicken noodle soup	58403010
12.5	Chicken noodle soup, home recipe	58403040
12.5	Chicken rice soup	58404010
12.5	Chicken soup with dumplings	58404520
12.5	Turkey noodle soup, home recipe	58406020
25.0	Turnover, chicken- or turkey-, and cheese-filled, no gravy	58126270
25.0	Turnover, chicken- or turkey-, and vegetable-filled, lower i	58126280
33.0	Chicken or turkey, potatoes, and vegetables (including carro	27341010
33.0	Chicken or turkey, potatoes, and vegetables (excluding carro	27341020
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341310
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341320
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341510
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341520
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343010
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343020
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343470
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343480
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343510
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343520
33.0	Chicken or turkey chow mein or chop suey with noodles	27343910
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343950
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343960
33.0	CHICKEN, NOODLES, VEG (NO CAR/DK GRN), CREAM SAUCE	27343980

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345010
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345020
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345210
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345220
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345310
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345320
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345410
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345420
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345440
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345520
33.0	Chicken or turkey pot pie	27347100
33.0	Chicken or turkey, dumplings, and vegetables (including carr	27347240
33.0	Chicken or turkey, dumplings, and vegetables (excluding carr	27347250
33.0	Chicken, fried, with potatoes, vegetable (frozen meal)	28140710
33.0	Chicken patty, or nuggets, boneless, breaded, potatoes, vege	28140720
33.0	Chicken patty, breaded, with tomato sauce and cheese, fettuc	28140730
33.0	Chicken patty, or nuggets, boneless, breaded, with pasta and	28140740
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28140810
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28141010
33.0	CHICKEN PATTY W/ VEGETABLES (DIET FROZEN MEAL)	28141060
33.0	CHICKEN TERIYAKI W/ RICE, VEGETABLE (FROZEN MEAL)	28141200
33.0	Chicken with rice-vegetable mixture (diet frozen meal)	28141250
33.0	Chicken with rice and vegetable, reduced fat and sodium (die	28141300
33.0	Chicken a la king with rice (frozen meal)	28141600
33.0	Chicken and vegetables in cream or white sauce (diet frozen	28141610
33.0	Chicken and vegetable entree with rice, Oriental (diet froze	28143020
33.0	Chicken and vegetable entree, oriental (diet frozen meal)	28143030
33.0	Chicken chow mein with rice (diet frozen meal)	28143040
33.0	Chicken with noodles and cheese sauce (diet frozen meal)	28143080
33.0	Chicken cacciatore with noodles (diet frozen meal)	28143110
33.0	Chicken and vegetable entree with noodles (frozen meal)	28143130
33.0	Chicken and vegetable entree with noodles (diet frozen meal)	28143150
33.0	Chicken in cream sauce with noodles and vegetable (frozen me	28143170

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken in butter sauce with potatoes and vegetable (diet fr	28143180
33.0	Chicken in soy-based sauce, rice and vegetables (frozen meal	28143200
33.0	Chicken in orange sauce with almond rice (diet frozen meal)	28143210
33.0	Chicken in barbecue sauce, with rice, vegetable and dessert,	28143220
33.0	Chicken and vegetable entree with noodles and cream sauce (f	28144100
33.0	Turkey dinner, NFS (frozen meal)	28145000
33.0	TURKEY W/ DRESSING, GRAVY, POTATO (FROZEN MEAL)	28145010
33.0	Turkey with dressing, gravy, vegetable and fruit (diet froze	28145100
33.0	Turkey with vegetable, stuffing (diet frozen meal)	28145110
33.0	Turkey with gravy, dressing, potatoes, vegetable (frozen mea	28145210
33.0	Turkey with gravy, dressing, potatoes, vegetable, dessert (f	28145610
33.0	Burrito with chicken, no beans	58100200
33.0	Burrito with chicken and beans	58100210
33.0	Burrito with chicken, beans, and cheese	58100220
33.0	Burrito with chicken and cheese	58100230
33.0	Burrito with chicken, NFS	58100240
33.0	Enchilada with chicken, tomato-based sauce	58100600
33.0	Enchilada with chicken, beans, and cheese, tomato- based sau	58100620
33.0	Enchilada with chicken and cheese, no beans, tomato- based s	58100630
33.0	Flauta with chicken	58101240
33.0	Soft taco with chicken, cheese, and lettuce	58101450
33.0	Soft taco with chicken, cheese, lettuce, tomato and sour cre	58101460
33.0	Taco or tostada with chicken or turkey, lettuce, tomato and	58101510
33.0	Taco or tostada with chicken, cheese, lettuce, tomato and sa	58101520
33.0	Nachos with chicken or turkey and cheese	58104250
33.0	Chimichanga with chicken and cheese	58104530
33.0	Fajita with chicken and vegetables	58105000
33.0	Cornmeal dressing with chicken or turkey and vegetables	58128120
33.0	Rice with chicken, Puerto Rican style (Arroz con Pollo)	58155110
50.0	Chicken or turkey and potatoes with gravy (mixture)	27241010
50.0	Chicken or turkey and noodles, no sauce (mixture)	27242000
50.0	Chicken or turkey and noodles with gravy (mixture)	27242200
50.0	Chicken or turkey and noodles with (mushroom) soup (mixture)	27242250

% Poultry in Food Item	Food Item Description	USDA Food Code
50.0	Chicken or turkey and noodles with cream or white sauce (mix	27242300
50.0	Chicken or turkey and noodles with cheese sauce (mixture)	27242310
50.0	Chicken or turkey and noodles, tomato-based sauce (mixture)	27242400
50.0	Chicken or turkey and rice, no sauce (mixture)	27243000
50.0	Chicken or turkey and rice with cream sauce (mixture)	27243300
50.0	Chicken or turkey and rice with (mushroom) soup (mixture)	27243400
50.0	Chicken or turkey and rice with tomato-based sauce (mixture)	27243500
50.0	Chicken or turkey and rice with soy-based sauce (mixture)	27243600
50.0	Chicken or turkey with dumplings (mixture)	27246100
50.0	Chicken or turkey with stuffing (mixture)	27246200
50.0	Chicken or turkey and vegetables (including carrots, broccol	27440110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27440120
50.0	Chicken or turkey and vegetables (including carrots, broccol	27442110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27442120
50.0	Chicken or turkey a la king with vegetables (including carro	27443110
50.0	Chicken or turkey a la king with vegetables (excluding carro	27443120
50.0	Chicken or turkey divan	27443150
50.0	Chicken or turkey and vegetables (including carrots, broccol	27445110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27445120
50.0	General Tso (General Gau) chicken	27445150
50.0	Moo Goo Gai Pan	27445180
50.0	Kung pao chicken	27445220
50.0	Almond chicken	27445250
50.0	Chicken or turkey chow mein or chop suey, no noodles	27446100
50.0	Chicken or turkey salad	27446200
50.0	Chicken or turkey salad with egg	27446220
50.0	Chicken or turkey garden salad (chicken and/or turkey, tomat	27446300
50.0	Chicken or turkey garden salad (chicken and/or turkey, other	27446310
50.0	Chicken or turkey and vegetables (including carrots, broccol	27446400
75.0	Meat loaf made with chicken or turkey	27246500
75.0	Chicken sandwich, with spread	27540110
75.0	Chicken barbecue sandwich	27540130
75.0	Chicken fillet (breaded, fried) sandwich	27540140

% Poultry in Food Item	Food Item Description	USDA Food Code
75.0	Chicken fillet (breaded, fried) sandwich with lettuce, tomat	27540150
75.0	Chicken patty sandwich, miniature, with spread	27540170
75.0	Chicken patty sandwich or biscuit	27540180
75.0	Chicken patty sandwich, with lettuce and spread	27540190
75.0	Fajita-style chicken sandwich with cheese, on pita bread, wi	27540200
75.0	Chicken patty sandwich with cheese, on wheat bun, with lettu	27540230
75.0	Chicken fillet, (broiled), sandwich, on whole wheat roll, wi	27540240
75.0	Chicken fillet, broiled, sandwich with cheese, on whole whea	27540250
75.0	Chicken fillet, broiled, sandwich, on oat bran bun, with let	27540260
75.0	Chicken fillet, broiled, sandwich, with lettuce, tomato, and	27540270
75.0	Chicken fillet, broiled, sandwich with cheese, on bun, with	27540280
100.0	Chicken, NS as to part and cooking method, NS as to skin eat	24100000
100.0	Chicken, NS as to part and cooking method, skin eaten	24100010
100.0	Chicken, NS as to part and cooking method, skin not eaten	24100020
100.0	CHICKEN, BONELESS, BROILED, NS PART, NS SKIN	24101000
100.0	CHICKEN, BONELESS, BROILED, NS PART, W/O SKIN	24101020
100.0	Chicken, NS as to part, roasted, broiled, or baked, NS as to	24102000
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin eat	24102010
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin not	24102020
100.0	Chicken, NS as to part, stewed, NS as to skin eaten	24103000
100.0	Chicken, NS as to part, stewed, skin eaten	24103010
100.0	Chicken, NS as to part, stewed, skin not eaten	24103020
100.0	Chicken, NS as to part, fried, no coating, NS as to skin eat	24104000
100.0	Chicken, NS as to part, fried, no coating, skin not eaten	24104020
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, NS SKIN	24105000
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, W/ SKIN	24105010
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, NS SKIN	24106000
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, W/ SKIN	24106010
100.0	CHICKEN,BONELESS,BREADD,BAKD/FRIED,W/O SKIN,NS COAT	24106040
100.0	CHICKEN,BONELESS,BREADD,BAKED/FRIED,W/O SKIN,W/COAT	24106050
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107000
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107010
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107020

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, NS as to part, coated, baked or fried, prepared ski	24107050
100.0	CHICKEN, W/ BONE, NFS	24110000
100.0	CHICKEN, W/ BONE, NS AS TO PART, ROASTED, W/ SKIN	24112010
100.0	CHICKEN,W/BONE,NS PART,BREADED,BAKD/FRIED, W/O SKIN	24116020
100.0	Chicken, breast, NS as to cooking method, NS as to skin eate	24120100
100.0	Chicken, breast, NS as to cooking method, skin eaten	24120110
100.0	Chicken, breast, NS as to cooking method, skin not eaten	24120120
100.0	CHICKEN, BREAST, BROILED, NS AS TO SKIN	24121100
100.0	CHICKEN, BREAST, BROILED, W/SKIN	24121110
100.0	CHICKEN, BREAST, BROILED, W/O SKIN	24121120
100.0	Chicken, breast, roasted, broiled, or baked, NS as to skin e	24122100
100.0	Chicken, breast, roasted, broiled, or baked, skin eaten	24122110
100.0	Chicken, breast, roasted, broiled, or baked, skin not eaten	24122120
100.0	Chicken, breast, stewed, NS as to skin eaten	24123100
100.0	Chicken, breast, stewed, skin eaten	24123110
100.0	Chicken, breast, stewed, skin not eaten	24123120
100.0	Chicken, breast, fried, no coating, NS as to skin eaten	24124100
100.0	Chicken, breast, fried, no coating, skin eaten	24124110
100.0	Chicken, breast, fried, no coating, skin not eaten	24124120
100.0	CHICKEN, BREAST, FLOURED,BAKED/FRIED, NS AS TO SKIN	24125100
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/ SKIN	24125110
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/O SKIN	24125120
100.0	CHICKEN,BREAST,FLOURED,BAKED/FRIED,W/O SKIN,NS COAT	24125140
100.0	CHICKEN, BREAST, BREADED,BAKED/FRIED, NS AS TO SKIN	24126100
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/ SKIN	24126110
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/O SKIN	24126120
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED, SKINLESS,W/COAT	24126150
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED,W/O SKIN,NO COAT	24126160
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127100
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127110
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127120
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127140
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127150

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127160
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130200
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130220
100.0	CHICKEN, LEG, BROILED, NS AS TO SKIN	24131200
100.0	CHICKEN, LEG, BROILED, W/ SKIN	24131210
100.0	CHICKEN, LEG, BROILED, W/O SKIN	24131220
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132200
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132210
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132220
100.0	Chicken, leg (drumstick and thigh), stewed, NS as to skin ea	24133200
100.0	Chicken, leg (drumstick and thigh), stewed, skin eaten	24133210
100.0	Chicken, leg (drumstick and thigh), stewed, skin not eaten	24133220
100.0	Chicken, leg (drumstick and thigh), fried, no coating, NS as	24134200
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134210
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134220
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, NS AS TO SKIN	24135200
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/ SKIN	24135210
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/O SKIN	24135220
100.0	CHICKEN, LEG, BREADED, BAKED/FRIED, W/ SKIN	24136210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137220
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137240
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137250
100.0	Chicken, drumstick, NS as to cooking method, NS as to skin e	24140200
100.0	Chicken, drumstick, NS as to cooking method, skin eaten	24140210
100.0	Chicken, drumstick, NS as to cooking method, skin not eaten	24140220
100.0	CHICKEN, DRUMSTICK, BROILED, NS AS TO SKIN	24141200
100.0	CHICKEN, DRUMSTICK, BROILED, W/ SKIN	24141210
100.0	CHICKEN, DRUMSTICK, BROILED, W/O SKIN	24141220
100.0	Chicken, drumstick, roasted, broiled, or baked, NS as to ski	24142200
100.0	Chicken, drumstick, roasted, broiled, or baked, skin eaten	24142210
100.0	Chicken, drumstick, roasted, broiled, or baked, skin not eat	24142220
100.0	Chicken, drumstick, stewed, NS as to skin eaten	24143200

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, drumstick, stewed, skin eaten	24143210
100.0	Chicken, drumstick, stewed, skin not eaten	24143220
100.0	Chicken, drumstick, fried, no coating, NS as to skin eaten	24144200
100.0	Chicken, drumstick, fried, no coating, skin eaten	24144210
100.0	Chicken, drumstick, fried, no coating, skin not eaten	24144220
100.0	CHICKEN, DRUMSTICK,FLOURED,BAKD/FRIED,NS AS TO SKIN	24145200
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/ SKIN	24145210
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/O SKIN	24145220
100.0	CHICKEN,DRUMSTICK,FLOURD,BAKD/FRID,W/O SKIN,W/ COAT	24145250
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/ SKIN	24146210
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/O SKIN	24146220
100.0	CHICKEN,DRUMSTICK,BREADED,BAKD/FRID,SKINLESS,W/COAT	24146250
100.0	CHICKEN,DRUMSTICK,BREADD,BAKD/FRID,W/O SKIN,NO COAT	24146260
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147200
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147210
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147220
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147240
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147250
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147260
100.0	Chicken, thigh, NS as to cooking method, NS as to skin eaten	24150200
100.0	Chicken, thigh, NS as to cooking method, skin eaten	24150210
100.0	Chicken, thigh, NS as to cooking method, skin not eaten	24150220
100.0	CHICKEN, THIGH, BROILED, NS AS TO SKIN	24151200
100.0	CHICKEN, THIGH, BROILED, W/ SKIN	24151210
100.0	CHICKEN, THIGH, BROILED, W/O SKIN	24151220
100.0	Chicken, thigh, roasted, broiled, or baked, NS as to skin e	24152200
100.0	Chicken, thigh, roasted, broiled, or baked, skin eaten	24152210
100.0	Chicken, thigh, roasted, broiled, or baked, skin not eaten	24152220
100.0	Chicken, thigh, stewed, NS as to skin eaten	24153200
100.0	Chicken, thigh, stewed, skin eaten	24153210
100.0	Chicken, thigh, stewed, skin not eaten	24153220
100.0	Chicken, thigh, fried, no coating, NS as to skin eaten	24154200
100.0	Chicken, thigh, fried, no coating, skin eaten	24154210

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, thigh, fried, no coating, skin not eaten	24154220
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, NS AS TO SKIN	24155200
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/ SKIN	24155210
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/O SKIN	24155220
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/ SKIN	24156210
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/O SKIN	24156220
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, SKINLESS, W/COATING	24156250
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/O SKIN, NO COAT	24156260
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157200
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157210
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157220
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, N	24157240
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157250
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157260
100.0	Chicken, wing, NS as to cooking method, NS as to skin eaten	24160100
100.0	Chicken, wing, NS as to cooking method, skin eaten	24160110
100.0	Chicken, wing, NS as to cooking method, skin not eaten	24160120
100.0	CHICKEN, WING, BROILED, W/ SKIN	24161110
100.0	CHICKEN, WING, BROILED, W/O SKIN	24161120
100.0	Chicken, wing, roasted, broiled, or baked, NS as to skin eat	24162100
100.0	Chicken, wing, roasted, broiled, or baked, skin eaten	24162110
100.0	Chicken, wing, roasted, broiled, or baked, skin not eaten	24162120
100.0	Chicken, wing, stewed, NS as to skin eaten	24163100
100.0	Chicken, wing, stewed, skin eaten	24163110
100.0	Chicken, wing, stewed, skin not eaten	24163120
100.0	Chicken, wing, fried, no coating, NS as to skin eaten	24164100
100.0	Chicken, wing, fried, no coating, skin eaten	24164110
100.0	Chicken, wing, fried, no coating, skin not eaten	24164120
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, NS AS TO SKIN	24165100
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/ SKIN	24165110
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/O SKIN	24165120
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/ SKIN	24166110
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/O SKIN	24166120

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, wing, coated, baked or fried, prepared with skin, N	24167100
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167110
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167120
100.0	Chicken, back	24170200
100.0	CHICKEN, BACK, ROASTED, W/O SKIN	24172220
100.0	CHICKEN, BACK, STEWED, NS AS TO SKIN	24173200
100.0	CHICKEN, BACK, STEWED, W/ SKIN	24173210
100.0	Chicken, neck or ribs	24180200
100.0	Chicken skin	24198440
100.0	Chicken feet	24198500
100.0	CHICKEN, CANNED, MEAT ONLY, LIGHT MEAT	24198550
100.0	Chicken, canned, meat only	24198570
100.0	CHICKEN ROLL, ROASTED, NS AS TO LIGHT OR DARK MEAT	24198640
100.0	Chicken patty, fillet, or tenders, breaded, cooked	24198700
100.0	Chicken, ground	24198720
100.0	Chicken nuggets	24198740
100.0	Chicken crackling, Puerto Rican style (Chicharron de pollo)	24198840
100.0	Turkey, NFS	24201000
100.0	Turkey, light meat, cooked, NS as to skin eaten	24201010
100.0	Turkey, light meat, cooked, skin not eaten	24201020
100.0	Turkey, light meat, cooked, skin eaten	24201030
100.0	Turkey, light meat, breaded, baked or fried, NS as to skin e	24201050
100.0	Turkey, light meat, breaded, baked or fried, skin not eaten	24201060
100.0	Turkey, light meat, roasted, NS as to skin eaten	24201110
100.0	Turkey, light meat, roasted, skin not eaten	24201120
100.0	Turkey, light meat, roasted, skin eaten	24201130
100.0	Turkey, dark meat, roasted, NS as to skin eaten	24201210
100.0	Turkey, dark meat, roasted, skin not eaten	24201220
100.0	Turkey, light and dark meat, roasted, NS as to skin eaten	24201310
100.0	Turkey, light and dark meat, roasted, skin not eaten	24201320
100.0	Turkey, light and dark meat, roasted, skin eaten	24201330
100.0	Turkey, light or dark meat, battered, fried, skin not eaten	24201360
100.0	Turkey, light or dark meat, stewed, NS as to skin eaten	24201400

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Turkey, light or dark meat, stewed, skin not eaten	24201410
100.0	Turkey, light or dark meat, smoked, cooked, NS as to skin ea	24201500
100.0	Turkey, light or dark meat, smoked, cooked, skin not eaten	24201520
100.0	Turkey, drumstick, cooked, skin not eaten	24202010
100.0	Turkey, drumstick, cooked, skin eaten	24202020
100.0	Turkey, drumstick, roasted, NS as to skin eaten	24202050
100.0	Turkey, drumstick, roasted, skin not eaten	24202060
100.0	Turkey, drumstick, roasted, skin eaten	24202070
100.0	Turkey, thigh, cooked, NS as to skin eaten	24202450
100.0	Turkey, thigh, cooked, skin eaten	24202460
100.0	Turkey, thigh, cooked, skin not eaten	24202500
100.0	Turkey, neck, cooked	24202600
100.0	Turkey, wing, cooked, NS as to skin eaten	24203000
100.0	Turkey, wing, cooked, skin not eaten	24203010
100.0	Turkey, wing, cooked, skin eaten	24203020
100.0	Turkey, rolled roast, light or dark meat, cooked	24204000
100.0	Turkey, canned	24206000
100.0	Turkey, ground	24207000
100.0	Turkey, nuggets	24208000
100.0	CHICKEN LIVER, BATTERED, FRIED	25110410
100.0	Chicken liver, braised	25110420
100.0	CHICKEN LIVER, FRIED OR SAUTEED, NO COATING	25110440
100.0	Chicken liver, fried	25110450
100.0	Liver paste or pate, chicken	25112200
100.0	Chicken or turkey cake, patty, or croquette	27246300

Table D. 6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, NS as to cut, cooked, NS as to fat eaten	21000100
100.0	Beef, NS as to cut, cooked, lean and fat eaten	21000110
100.0	Beef, NS as to cut, cooked, lean only eaten	21000120
100.0	Steak, NS as to type of meat, cooked, NS as to fat eaten	21001000
100.0	Steak, NS as to type of meat, cooked, lean and fat eaten	21001010
100.0	Steak, NS as to type of meat, cooked, lean only eaten	21001020
100.0	Beef, pickled	21002000
100.0	Beef, NS as to cut, fried, NS to fat eaten	21003000
100.0	Beef steak, NS as to cooking method, NS as to fat eaten	21101000
100.0	Beef steak, NS as to cooking method, lean and fat eaten	21101010
100.0	Beef steak, NS as to cooking method, lean only eaten	21101020
100.0	Beef steak, broiled or baked, NS as to fat eaten	21101110
100.0	Beef steak, broiled or baked, lean and fat eaten	21101120
100.0	Beef steak, broiled or baked, lean only eaten	21101130
100.0	Beef steak, fried, NS as to fat eaten	21102110
100.0	Beef steak, fried, lean and fat eaten	21102120
100.0	Beef steak, fried, lean only eaten	21102130
100.0	Beef steak, breaded or floured, baked or fried, NS as to fat	21103110
100.0	Beef steak, breaded or floured, baked or fried, lean and fat	21103120
100.0	Beef steak, breaded or floured, baked or fried, lean only ea	21103130
100.0	Beef steak, battered, fried, NS as to fat eaten	21104110
100.0	Beef steak, battered, fried, lean and fat eaten	21104120
100.0	Beef steak, battered, fried, lean only eaten	21104130
100.0	Beef steak, braised, NS as to fat eaten	21105110
100.0	Beef steak, braised, lean and fat eaten	21105120
100.0	Beef steak, braised, lean only eaten	21105130
100.0	Beef, oxtails, cooked	21301000
100.0	Beef, neck bones, cooked	21302000
100.0	Beef, shortribs, cooked, NS as to fat eaten	21304000
100.0	Beef, shortribs, cooked, lean and fat eaten	21304110
100.0	Beef, shortribs, cooked, lean only eaten	21304120

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, shortribs, barbecued, with sauce, NS as to fat eaten	21304200
100.0	Beef, shortribs, barbecued, with sauce, lean and fat eaten	21304210
100.0	Beef, shortribs, barbecued, with sauce, lean only eaten	21304220
100.0	Beef, cow head, cooked	21305000
100.0	Beef, roast, roasted, NS as to fat eaten	21401000
100.0	Beef, roast, roasted, lean and fat eaten	21401110
100.0	Beef, roast, roasted, lean only eaten	21401120
100.0	Beef, roast, canned	21401400
100.0	Beef, pot roast, braised or boiled, NS as to fat eaten	21407000
100.0	Beef, pot roast, braised or boiled, lean and fat eaten	21407110
100.0	Beef, pot roast, braised or boiled, lean only eaten	21407120
100.0	Beef, stew meat, cooked, NS as to fat eaten	21410000
100.0	Beef, stew meat, cooked, lean and fat eaten	21410110
100.0	Beef, stew meat, cooked, lean only eaten	21410120
100.0	Beef brisket, cooked, NS as to fat eaten	21417100
100.0	Beef brisket, cooked, lean and fat eaten	21417110
100.0	Beef brisket, cooked, lean only eaten	21417120
100.0	Beef, sandwich steak (flaked, formed, thinly sliced)	21420100
100.0	Ground beef or patty, cooked, NS as to regular, lean, or ext	21500100
100.0	Ground beef, meatballs, meat only, cooked, NS as to regular,	21500110
100.0	Ground beef or patty, breaded, cooked	21500200
100.0	Ground beef, regular, cooked	21501000
100.0	Ground beef, lean, cooked	21501200
100.0	Ground beef, extra lean, cooked	21501300
100.0	Beef, bacon, cooked	21601000
100.0	Beef, bacon, cooked, lean only eaten	21601250
100.0	Beef, dried, chipped, uncooked	21602000
100.0	Beef jerky	21602100
100.0	Beef, pastrami (beef, smoked, spiced)	21603000
100.0	Beef, baby food, strained	21701010
100.0	Beef liver, braised	25110120
100.0	Beef liver, fried	25110140
100.0	Beef sausage, NFS	25220100

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef sausage, fresh, bulk, patty or link, cooked	25220140
66.0	Beef with tomato-based sauce (mixture)	27111000
66.0	Spaghetti sauce with beef or meat other than lamb or mutton,	27111050
66.0	Beef goulash	27111100
66.0	Mexican style beef stew, no potatoes, tomato-based sauce (mi	27111300
66.0	Mexican style beef stew, no potatoes, with chili peppers, to	27111310
66.0	Beef sloppy joe (no bun)	27111500
66.0	Beef with gravy (mixture)	27112000
66.0	Salisbury steak with gravy (mixture)	27112010
66.0	Beef stroganoff	27113100
66.0	Creamed chipped or dried beef	27113200
66.0	Beef with (mushroom) soup (mixture)	27114000
66.0	Beef with soy-based sauce (mixture)	27115000
66.0	Steak teriyaki with sauce (mixture)	27115100
66.0	Beef with barbecue sauce (mixture)	27116200
66.0	Beef with sweet and sour sauce (mixture)	27116300
66.0	Stewed, seasoned, ground beef, Mexican style (Picadillo de c	27116350
66.0	Stewed seasoned ground beef, Puerto Rican style (Picadillo g	27118120
33.0	Beef and potatoes, no sauce (mixture)	27211000
33.0	Beef stew with potatoes, tomato-based sauce (mixture)	27211100
33.0	Mexican style beef stew with potatoes, tomato-based sauce (m	27211110
33.0	Beef goulash with potatoes	27211150
33.0	Beef and potatoes with cream sauce, white sauce or mushroom	27211190
33.0	Beef stew with potatoes, gravy	27211200
33.0	Beef and potatoes with cheese sauce (mixture)	27211500
33.0	Stewed, seasoned, ground beef with potatoes, Mexican style (27211550
33.0	Beef and noodles, no sauce (mixture)	27212000
33.0	Beef and macaroni with cheese sauce (mixture)	27212050
33.0	Beef and noodles with tomato-based sauce (mixture)	27212100
33.0	Chili con carne with beans and macaroni	27212120
33.0	Beef goulash with noodles	27212150
33.0	Beef and noodles with gravy (mixture)	27212200
33.0	Beef and noodles with cream or white sauce (mixture)	27212300

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef stroganoff with noodles	27212350
33.0	Beef and noodles with (mushroom) soup (mixture)	27212400
33.0	Beef and rice, no sauce (mixture)	27213000
33.0	Beef and rice with tomato-based sauce (mixture)	27213100
33.0	Porcupine balls with tomato-based sauce (mixture)	27213120
33.0	Chili con carne with beans and rice	27213150
33.0	Beef and rice with gravy (mixture)	27213200
33.0	Beef and rice with cream sauce (mixture)	27213300
33.0	Beef and rice with soy-based sauce (mixture)	27213500
66.0	Meat loaf made with beef	27214100
66.0	Meat loaf made with beef, with tomato-based sauce	27214110
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
50.0	Meat loaf made with beef and pork	27260080
33.0	Meat loaf made with beef, veal and pork	27260090
66.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311110
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311310
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311320
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311410
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311420
33.0	Shepherd's pie with beef	27311510
33.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311610
33.0	Beef, potatoes, and vegetables (excluding carrots, broccoli,	27311620
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313010
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313020
33.0	Beef chow mein or chop suey with noodles	27313110
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313150
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313160
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313210
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313220
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313410
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315010

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315020
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315210
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315220
33.0	Stuffed cabbage rolls with beef and rice	27315250
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315310
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315410
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315510
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315520
33.0	Beef pot pie	27317010
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27410210
50.0	Beef and vegetables (excluding carrots, broccoli, and dark-g	27410220
50.0	Beef shish kabob with vegetables, excluding potatoes	27410250
50.0	Beef with vegetables (including carrots, broccoli, and/or da	27411100
50.0	Swiss steak	27411120
50.0	Beef rolls, stuffed with vegetables or meat mixture, tomato	27411150
50.0	Beef with vegetables (excluding carrots, broccoli, and dark	27411200
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27415100
50.0	Beef, tofu, and vegetables (including carrots, broccoli, and	27415120
50.0	Beef chow mein or chop suey, no noodles	27415150
100.0	Pepper steak	27416150
66.0	Beef steak with onions, Puerto Rican style (mixture) (Biftec	27418410
100.0	Liver, beef or calves, and onions	27460750
66.0	Beef barbecue sandwich or Sloppy Joe, on bun	27510110
66.0	Cheeseburger, plain, on bun	27510210
66.0	Cheeseburger, with mayonnaise or salad dressing, on bun	27510220
66.0	Cheeseburger, with mayonnaise or salad dressing and tomatoes	27510230
66.0	Cheeseburger, 1/4 lb meat, plain, on bun	27510240
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510250
66.0	Cheeseburger, 1/4 lb meat, with mushrooms in sauce, on bun	27510260
66.0	Double cheeseburger (2 patties), plain, on bun	27510270
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510280

% Beef in Food Item	Food Item Description	USDA food code value
66.0	Double cheeseburger (2 patties), plain, on double-decker bun	27510290
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510300
66.0	Cheeseburger with tomato and/or catsup, on bun	27510310
66.0	Cheeseburger, 1 oz meat, plain, on miniature bun	27510311
66.0	Cheeseburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510320
66.0	Double cheeseburger (2 patties), with tomato and/or catsup,	27510330
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510340
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510350
66.0	Cheeseburger with mayonnaise or salad dressing, tomato and b	27510360
66.0	Double cheeseburger (2 patties, 1/4 lb meat each), with mayo	27510370
66.0	Triple cheeseburger (3 patties, 1/4 lb meat each), with mayo	27510380
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), on	27510390
66.0	Bacon cheeseburger, 1/4 lb meat, with tomato and/or catsup,	27510400
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), wit	27510430
66.0	Bacon cheeseburger, 1/4 lb meat, with mayonnaise or salad dr	27510440
66.0	Hamburger, plain, on bun	27510500
66.0	Hamburger, with tomato and/or catsup, on bun	27510510
66.0	Hamburger, with mayonnaise or salad dressing and tomatoes, o	27510520
66.0	Hamburger, 1/4 lb meat, plain, on bun	27510530
66.0	Double hamburger (2 patties), with tomato and/or catsup, on	27510540
66.0	Hamburger, 1/4 lb meat, with mayonnaise or salad dressing an	27510560
66.0	Hamburger, with mayonnaise or salad dressing, on bun	27510590
66.0	Hamburger, 1 oz meat, plain, on miniature bun	27510600
66.0	Hamburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510620
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510660
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510670
66.0	Double hamburger (2 patties, 1/4 lb meat each), with tomato	27510680
66.0	Double hamburger (2 patties, 1/4 lb meat each), with mayonna	27510690
66.0	Meatball and spaghetti sauce submarine sandwich	27510700
66.0	Roast beef sandwich	27513010
66.0	Roast beef submarine sandwich, with lettuce, tomato and spre	27513040
66.0	Roast beef sandwich with cheese	27513050

% Beef in Food Item	Food Item Description	USDA food code value
66.0	Roast beef sandwich with bacon and cheese sauce	27513060
66.0	Steak submarine sandwich with lettuce and tomato	27515000
66.0	Steak sandwich, plain, on roll	27515010
50.0	Beef dinner, NFS (frozen meal)	28110000
50.0	Beef with potatoes (frozen meal, large meat portion)	28110120
50.0	Beef with vegetable (diet frozen meal)	28110150
33.0	Sirloin, chopped, with gravy, mashed potatoes, vegetable (fr	28110220
33.0	Sirloin beef with gravy, potatoes, vegetable (frozen meal)	28110270
33.0	Salisbury steak dinner, NFS (frozen meal)	28110300
33.0	Salisbury steak with gravy, potatoes, vegetable (frozen meal	28110310
33.0	Salisbury steak with gravy, whipped potatoes, vegetable, des	28110330
33.0	Salisbury steak with gravy, potatoes, vegetable, dessert (fr	28110350
33.0	Salisbury steak with gravy, macaroni and cheese, vegetable (28110370
33.0	Salisbury steak with gravy, macaroni and cheese (frozen meal	28110380
33.0	Salisbury steak, potatoes, vegetable, dessert (diet frozen m	28110390
33.0	Beef, sliced, with gravy, potatoes, vegetable (frozen meal)	28110510
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
25.0	Chili beef soup	28310210
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
25.0	Beef and rice noodle soup, Oriental style (Vietnamese Pho Bo	28310330
25.0	Beef and rice soup, Puerto Rican style	28310420
25.0	Pepperpot (tripe) soup	28311010
25.0	Beef vegetable soup with potato, stew type	28315100
25.0	Beef vegetable soup with noodles, stew type, chunky style	28315120
25.0	Beef vegetable soup with rice, stew type, chunky style	28315130
25.0	Beef vegetable soup, Mexican style (Sopa / caldo de Res)	28315140
33.0	Burrito with beef, no beans	58100100
33.0	Burrito with beef and beans	58100110
33.0	Burrito with beef, beans, and cheese	58100120
33.0	Burrito with beef and cheese, no beans	58100130
33.0	Burrito with beef, beans, cheese, and sour cream	58100140
33.0	Burrito with beef and potato, no beans	58100150

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Enchilada with beef, no beans	58100400
33.0	Enchilada with beef and beans	58100510
33.0	Enchilada with beef, beans, and cheese	58100520
33.0	Enchilada with beef and cheese, no beans	58100530
33.0	Flauta with beef	58101230
33.0	Taco or tostada with beef, cheese and lettuce	58101300
33.0	Taco or tostada with beef, lettuce, tomato and salsa	58101310
33.0	Taco or tostada with beef, cheese, lettuce, tomato and salsa	58101320
33.0	Soft taco with beef, cheese, lettuce, tomato and sour cream	58101350
33.0	Soft taco with beef, cheese, and lettuce	58101400
33.0	Mexican casserole made with ground beef, tomato sauce, cheese	58101830
33.0	Taco or tostada salad with beef and cheese, corn chips	58101910
33.0	Taco or tostada salad with beef, beans and cheese, fried flour	58101930
12.5	Tamale casserole with meat	58103310
33.0	Nachos with beef, beans, cheese, and sour cream	58104080
33.0	Nachos with beef, beans, cheese, tomatoes, sour cream and onion	58104180
33.0	Chimichanga with beef and tomato	58104450
33.0	Chimichanga, NFS	58104490
33.0	Chimichanga with beef, beans, lettuce and tomato	58104500
33.0	Chimichanga with beef, cheese, lettuce and tomato	58104510
12.5	Quesadilla with meat and cheese	58104730
33.0	Fajita with beef and vegetables	58105050
25.0	Macaroni or noodles with cheese and beef	58145130
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Barley soup	58401010
12.5	Beef noodle soup	58402010
12.5	Beef dumpling soup	58402020
12.5	Beef rice soup	58402030
12.5	Beef noodle soup, home recipe	58402100

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
25.0	Brunswick stew	27360100
25.0	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
25.0	Meat and corn hominy soup, Mexican style (Pozole)	28315150
25.0	Pork and rice soup, stew type, chunky style	28320110
25.0	Pork, vegetable soup with potatoes, stew type	28320150
25.0	Pork with vegetable (excluding carrots, broccoli and/or dark	28320300
33.0	Meat loaf made with beef, veal and pork	27260090
33.0	Ham or pork, noodles, and vegetables (including carrots, bro	27320070
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320110
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320210
33.0	Pork chow mein or chop suey with noodles	27320310
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420060
33.0	Greens with ham or pork (mixture)	27420080
33.0	Moo Shu (Mu Shi) Pork, without Chinese pancake	27420160
33.0	Pork and vegetables (excluding carrots, broccoli, and dark-g	27420350
33.0	Pork chow mein or chop suey, no noodles	27420390
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420410
33.0	Sausage and vegetables (including carrots, broccoli, and/or	27420450
33.0	Sausage and vegetables (excluding carrots, broccoli, and dar	27420460
33.0	Sausage and peppers, no sauce (mixture)	27420470
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420500
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420510

% Pork in Food Item	Food Item Description	USDA food code value
33.0	Burrito with pork and beans	58100180
50.0	Meat loaf made with beef and pork	27260080
50.0	Ham or pork salad	27420020
66.0	Pork and rice with tomato-based sauce (mixture)	27220110
66.0	Sausage and rice with tomato-based sauce (mixture)	27220120
66.0	Sausage and rice with (mushroom) soup (mixture)	27220150
66.0	Sausage and noodles with cream or white sauce (mixture)	27220190
66.0	Ham or pork and rice, no sauce (mixture)	27220310
66.0	Ham or pork and potatoes with gravy (mixture)	27220510
66.0	Stewed pig's feet, Puerto Rican style (Patitas de cerdo guis	27221100
66.0	Mexican style pork stew, with potatoes, tomato-based sauce (27221150
66.0	Pork sandwich, on white roll, with onions, dill pickles and	27520500
100.0	Pork, NS as to cut, cooked, NS as to fat eaten	22000100
100.0	Pork, NS as to cut, cooked, lean and fat eaten	22000110
100.0	Pork, NS as to cut, cooked, lean only eaten	22000120
100.0	Pork, NS as to cut, fried, NS as to fat eaten	22000200
100.0	Pork, NS as to cut, fried, lean and fat eaten	22000210
100.0	Pork, NS as to cut, fried, lean only eaten	22000220
100.0	Pork, NS as to cut, breaded or floured, fried, NS as to fat	22000300
100.0	Pork, NS as to cut, breaded or floured, fried, lean and fat	22000310
100.0	Pork, NS as to cut, breaded or floured, fried, lean only eat	22000320
100.0	Pork, pickled, NS as to cut	22001000
100.0	Pork, ground or patty, cooked	22002000
100.0	Pork, ground or patty, breaded, cooked	22002100
100.0	Pork jerky	22002800
100.0	Pork chop, NS as to cooking method, NS as to fat eaten	22101000
100.0	Pork chop, NS as to cooking method, lean and fat eaten	22101010
100.0	Pork chop, NS as to cooking method, lean only eaten	22101020
100.0	Pork chop, broiled or baked, NS as to fat eaten	22101100
100.0	Pork chop, broiled or baked, lean and fat eaten	22101110
100.0	Pork chop, broiled or baked, lean only eaten	22101120
100.0	Pork chop, breaded or floured, broiled or baked, lean and fa	22101140
100.0	Pork chop, breaded or floured, broiled or baked, lean only e	22101150

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork chop, fried, NS as to fat eaten	22101200
100.0	Pork chop, fried, lean and fat eaten	22101210
100.0	Pork chop, fried, lean only eaten	22101220
100.0	Pork chop, breaded or floured, fried, NS as to fat eaten	22101300
100.0	Pork chop, breaded or floured, fried, lean and fat eaten	22101310
100.0	Pork chop, breaded or floured, fried, lean only eaten	22101320
100.0	Pork chop, battered, fried, NS as to fat eaten	22101400
100.0	Pork chop, battered, fried, lean and fat eaten	22101410
100.0	Pork chop, battered, fried, lean only eaten	22101420
100.0	Pork chop, stewed, NS as to fat eaten	22101500
100.0	Pork chop, stewed, lean and fat eaten	22101510
100.0	Pork chop, stewed, lean only eaten	22101520
100.0	Pork chop, smoked or cured, cooked, lean and fat eaten	22107010
100.0	Pork chop, smoked or cured, cooked, lean only eaten	22107020
100.0	Pork steak or cutlet, NS as to cooking method, NS as to fat	22201000
100.0	Pork steak or cutlet, NS as to cooking method, lean and fat	22201010
100.0	Pork steak or cutlet, NS as to cooking method, lean only eat	22201020
100.0	Pork steak or cutlet, battered, fried, NS as to fat eaten	22201050
100.0	Pork steak or cutlet, battered, fried, lean and fat eaten	22201060
100.0	Pork steak or cutlet, battered, fried, lean only eaten	22201070
100.0	Pork steak or cutlet, broiled or baked, NS as to fat eaten	22201100
100.0	Pork steak or cutlet, broiled or baked, lean and fat eaten	22201110
100.0	Pork steak or cutlet, broiled or baked, lean only eaten	22201120
100.0	Pork steak or cutlet, fried, NS as to fat eaten	22201200
100.0	Pork steak or cutlet, fried, lean and fat eaten	22201210
100.0	Pork steak or cutlet, fried, lean only eaten	22201220
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201310
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201320
100.0	Pork steak or cutlet, breaded or floured, fried, NS as to fa	22201400
100.0	Pork steak or cutlet, breaded or floured, fried, lean and fa	22201410
100.0	Pork steak or cutlet, breaded or floured, fried, lean only e	22201420
100.0	Pork, tenderloin, cooked, NS as to cooking method	22210300
100.0	Pork, tenderloin, breaded, fried	22210310

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork, tenderloin, braised	22210350
100.0	Pork, tenderloin, baked	22210400
100.0	Pork roast, NS as to cut, cooked, NS as to fat eaten	22400100
100.0	Pork roast, NS as to cut, cooked, lean and fat eaten	22400110
100.0	Pork roast, NS as to cut, cooked, lean only eaten	22400120
100.0	Pork roast, loin, cooked, NS as to fat eaten	22401000
100.0	Pork roast, loin, cooked, lean and fat eaten	22401010
100.0	Pork roast, loin, cooked, lean only eaten	22401020
100.0	Pork roast, shoulder, cooked, lean only eaten	22411020
100.0	Pork roast, smoked or cured, cooked, NS as to fat eaten	22421000
100.0	Pork roast, smoked or cured, cooked, lean and fat eaten	22421010
100.0	Pork roast, smoked or cured, cooked, lean only eaten	22421020
100.0	Canadian bacon, cooked	22501010
100.0	Bacon, NS as to type of meat, cooked	22600100
100.0	Pork bacon, NS as to fresh, smoked or cured, cooked	22600200
100.0	Pork bacon, smoked or cured, cooked	22601000
100.0	Pork bacon, smoked or cured, cooked, lean only eaten	22601020
100.0	Bacon or side pork, fresh, cooked	22601040
100.0	Pork bacon, smoked or cured, lower sodium	22602010
100.0	Pork bacon, formed, lean meat added, cooked	22605010
100.0	Salt pork, cooked	22621000
100.0	Fat back, cooked	22621100
100.0	Pork, spareribs, cooked, NS as to fat eaten	22701000
100.0	Pork, spareribs, cooked, lean and fat eaten	22701010
100.0	Pork, spareribs, cooked, lean only eaten	22701020
100.0	Pork, spareribs, barbecued, with sauce, NS as to fat eaten	22701030
100.0	Pork, spareribs, barbecued, with sauce, lean and fat eaten	22701040
100.0	Pork, spareribs, barbecued, with sauce, lean only eaten	22701050
100.0	Pork, cracklings, cooked	22704010
100.0	Pork ears, tail, head, snout, miscellaneous parts, cooked	22705010
100.0	Pork, neck bones, cooked	22706010
100.0	Pork, pig's feet, cooked	22707010
100.0	Pork, pig's feet, pickled	22707020

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork, pig's hocks, cooked	22708010
100.0	Pork skin, rinds, deep-fried	22709010
100.0	Pork skin, boiled	22709110
100.0	PORK LIVER, BREADED, FRIED	25110340
100.0	Pork sausage, fresh, bulk, patty or link, cooked	25221410

Table D.8 Food Codes for Egg Items

% Eggs in Food Item	Food Item Description	USDA Food Code
25	Fried egg sandwich	32201000
25	Egg, cheese, and ham on English muffin	32202010
25	Egg, cheese, and ham on biscuit	32202020
25	Egg, cheese and ham on bagel	32202025
25	Egg, cheese, and sausage on English muffin	32202030
25	Egg, cheese, and beef on English Muffin	32202040
25	Egg, cheese, and steak on bagel	32202045
25	Egg, cheese, and sausage on biscuit	32202050
25	Egg, cheese, and sausage griddle cake sandwich	32202055
25	Egg and sausage on biscuit	32202060
25	Egg, cheese, and bacon on biscuit	32202070
25	Egg, cheese, and bacon griddle cake sandwich	32202075
25	Egg, cheese, and bacon on English muffin	32202080
25	Egg, cheese and bacon on bagel	32202085
25	Egg and bacon on biscuit	32202090
25	Egg and ham on biscuit	32202110
25	Egg, cheese and sausage on bagel	32202120
25	Egg and cheese on biscuit	32202200
25	Egg drop soup	32300100
25	Garlic egg soup, Puerto Rican style (Sopa de ajo)	32301100
25	Burrito with eggs, sausage, cheese and vegetables	58100340
25	Burrito with eggs and cheese, no beans	58100350
25	Croissant sandwich with sausage and egg	58127270
25	Croissant sandwich with ham, egg, and cheese	58127310
25	Croissant sandwich with sausage, egg, and cheese	58127330
25	Croissant sandwich with bacon, egg, and cheese	58127350
33	Egg dessert, custard-like, made with water and sugar, Puerto	32120100
66	Egg foo yung (young), NFS	32105200
66	Chicken egg foo yung (young)	32105210
66	Pork egg foo yung (young)	32105220
66	Shrimp egg foo yung (young)	32105230
75	Egg, Benedict	32101500

% Eggs in Food Item	Food Item Description	USDA Food Code
75	Egg, deviled	32102000
75	Egg salad	32103000
100	Egg, whole, raw	31101010
100	Egg, whole, cooked, NS as to cooking method	31102000
100	Egg, whole, boiled	31103000
100	Egg, whole, poached	31104000
100	Egg, whole, fried	31105000
100	Egg, whole, fried without fat	31105010
100	Egg, whole, baked, fat not added in cooking	31106010
100	Egg, whole, baked, fat added in cooking	31106020
100	Egg, whole, pickled	31107000
100	Egg, white only, cooked	31109010
100	Egg, yolk only, raw	31110010
100	Egg, yolk only, cooked	31111010
100	Egg, creamed	32101000
100	Egg omelet or scrambled egg, NS as to fat added in cooking	32104900
100	Egg omelet or scrambled egg, fat not added in cooking	32104950
100	Egg omelet or scrambled egg, fat added in cooking	32105000
100	Egg omelet or scrambled egg, with cheese	32105010
100	Egg omelet or scrambled egg, with fish	32105020
100	Egg omelet or scrambled egg, with ham or bacon	32105030
100	Egg omelet or scrambled egg, with dark-green vegetables	32105040
100	Egg omelet or scrambled egg, with vegetables other than dark	32105050
100	Egg omelet or scrambled egg, with peppers, onion, and ham	32105060
100	Egg omelet or scrambled egg, with mushrooms	32105070
100	Egg omelet or scrambled egg, with cheese and ham or bacon	32105080
100	Egg omelet or scrambled egg, with cheese, ham or bacon, and	32105085
100	Egg omelet or scrambled egg, with potatoes and/or onions (To	32105100
100	Egg omelet or scrambled egg, with beef	32105110
100	Egg omelet or scrambled egg, with sausage and cheese	32105121
100	Egg omelet or scrambled egg, with sausage	32105122
100	Egg omelet or scrambled egg, with hot dogs	32105125
100	Egg omelet or scrambled egg, with onions, peppers, tomatoes,	32105130

% Eggs in Food Item	Food Item Description	USDA Food Code
100	Egg omelet or scrambled egg, with chorizo	32105160
100	Egg omelet or scrambled egg with chicken	32105170
100	Huevos rancheros	32105180
100	Meringues	32401000

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
50	Cafe con leche prepared with sugar	11561010
50	Ice cream sandwich	13120500
50	Ice cream cookie sandwich	13120550
50	Ice cream cone with nuts, flavors other than chocolate	13120700
50	Ice cream cone, chocolate covered, with nuts, flavors other	13120710
50	Ice cream cone, chocolate covered or dipped, flavors other t	13120720
50	Ice cream cone, no topping, flavors other than chocolate	13120730
50	Ice cream cone, no topping, NS as to flavor	13120740
50	Ice cream cone with nuts, chocolate ice cream	13120750
50	Ice cream cone, chocolate covered or dipped, chocolate ice c	13120760
50	Ice cream cone, no topping, chocolate ice cream	13120770
50	Ice cream cone, chocolate covered, with nuts, chocolate ice	13120780
50	Ice cream sundae cone	13120790
50	Ice cream soda, flavors other than chocolate	13120800
50	Ice cream sundae, fruit topping, with whipped cream	13121100
50	Ice cream sundae, chocolate or fudge topping, with whipped c	13121300
50	Ice cream pie, no crust	13122100
50	Pudding, bread	13210110
50	Pudding, Mexican bread (Capirotada)	13210180
50	Cheese sandwich	14640000
50	Cheese sandwich, grilled	14640100
50	Cheese, nuggets or pieces, breaded, baked, or fried	14660200
75	Pudding, with fruit and vanilla wafers	13241000
100	Milk, NFS	11100000
100	Milk, cow's, fluid, whole	11111000
100	Milk, calcium fortified, cow's, fluid, whole	11111150
100	Milk, cow's, fluid, other than whole, NS as to 2%, 1%, or sk	11112000
100	Milk, cow's, fluid, 2% fat	11112110
100	Milk, cow's, fluid, acidophilus, 1% fat	11112120
100	Milk, cow's, fluid, acidophilus, 2% fat	11112130
100	Milk, cow's, fluid, 1% fat	11112210
100	Milk, cow's, fluid, skim or nonfat, 0.5% or less butterfat	11113000
100	Milk, cow's, fluid, lactose reduced, 1% fat	11114300

% Milk in Food Item	Food Item Description	USDA Food Code
100	Milk, cow's, fluid, lactose reduced, nonfat	11114320
100	Milk, cow's, fluid, lactose reduced, 2% fat	11114330
100	Milk, cow's, fluid, lactose reduced, whole	11114350
100	Buttermilk, fluid, nonfat	11115000
100	Buttermilk, fluid, 1% fat	11115100
100	Buttermilk, fluid, 2% fat	11115200
100	Milk, goat's, fluid, whole	11116000
100	Yogurt, NS as to type of milk or flavor	11410000
100	Yogurt, plain, NS as to type of milk	11411010
100	Yogurt, plain, whole milk	11411100
100	Yogurt, plain, lowfat milk	11411200
100	Yogurt, plain, nonfat milk	11411300
100	Yogurt, vanilla, lemon, or coffee flavor, NS as to type of m	11420000
100	Yogurt, vanilla, lemon, or coffee flavor, whole milk	11421000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, lowfat milk	11422000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11423000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11424000
100	Yogurt, chocolate, NS as to type of milk	11425000
100	Yogurt, fruit variety, NS as to type of milk	11430000
100	Yogurt, fruit variety, whole milk	11431000
100	Yogurt, fruit variety, lowfat milk	11432000
100	Yogurt, fruit variety, lowfat milk, sweetened with low-calor	11432500
100	Yogurt, fruit variety, nonfat milk	11433000
100	Yogurt, fruit variety, nonfat milk, sweetened with low-calor	11433500
100	Yogurt, fruit and nuts, lowfat milk	11445000
100	Yogurt, frozen, NS as to flavor, NS as to type of milk	11459990
100	Yogurt, frozen, flavors other than chocolate, NS as to type	11460000
100	Yogurt, frozen, chocolate, NS as to type of milk	11460100
100	Yogurt, frozen, NS as to flavor, lowfat milk	11460150
100	Yogurt, frozen, chocolate, lowfat milk	11460160
100	Yogurt, frozen, flavors other than chocolate, lowfat milk	11460170
100	Yogurt, frozen, NS as to flavor, nonfat milk	11460190
100	Yogurt, frozen, chocolate, nonfat milk	11460200
100	Yogurt, frozen, flavors other than chocolate, nonfat milk	11460300

% Milk in Food Item	Food Item Description	USDA Food Code
100	Yogurt, frozen, chocolate, nonfat milk, with low-calorie swe	11460400
100	Yogurt, frozen, flavors other than chocolate, nonfat milk, w	11460410
100	Yogurt, frozen, flavors other than chocolate, whole milk	11460440
100	Yogurt, frozen, cone, chocolate	11461250
100	Yogurt, frozen, cone, flavors other than chocolate	11461260
100	Yogurt, frozen, cone, flavors other than chocolate, lowfat m	11461270
100	Yogurt, frozen, cone, chocolate, lowfat milk	11461280
100	Milk, chocolate, NFS	11511000
100	Milk, chocolate, whole milk-based	11511100
100	Milk, chocolate, reduced fat milk-based, 2% (formerly "lowfa	11511200
100	Milk, chocolate, skim milk-based	11511300
100	Milk, chocolate, lowfat milk-based	11511400
100	Cocoa, hot chocolate, not from dry mix, made with whole milk	11512000
100	Cocoa and sugar mixture, milk added, NS as to type of milk	11513000
100	Cocoa and sugar mixture, whole milk added	11513100
100	Cocoa and sugar mixture, reduced fat milk added	11513150
100	Cocoa and sugar mixture, lowfat milk added	11513200
100	Cocoa and sugar mixture, skim milk added	11513300
100	Chocolate syrup, milk added, NS as to type of milk	11513400
100	Chocolate syrup, whole milk added	11513500
100	Chocolate syrup, reduced fat milk added	11513550
100	Chocolate syrup, lowfat milk added	11513600
100	Chocolate syrup, skim milk added	11513700
100	Cocoa, whey, and low-calorie sweetener mixture, lowfat milk	11516000
100	Milk beverage, made with whole milk, flavors other than choc	11519000
100	Milk, flavors other than chocolate, whole milk-based	11519050
100	Milk, malted, unfortified, NS as to flavor, made with milk	11520000
100	Milk, malted, unfortified, chocolate, made with milk	11521000
100	Milk, malted, unfortified, natural flavor, made with milk	11522000
100	Milk, malted, fortified, chocolate, made with milk	11526000
100	Milk, malted, fortified, NS as to flavor, made with milk	11527000
100	Eggnog, made with whole milk	11531000
100	Eggnog, made with 2% reduced fat milk (formerly eggnog, made	11531500

% Milk in Food Item	Food Item Description	USDA Food Code
100	Milk shake, homemade or fountain-type, NS as to flavor	11541100
100	Milk shake, homemade or fountain-type, chocolate	11541110
100	Milk shake, homemade or fountain-type, flavors other than ch	11541120
100	Milk shake with malt	11541400
100	Milk shake, made with skim milk, chocolate	11541500
100	Milk shake, made with skim milk, flavors other than chocolat	11541510
100	Milk fruit drink	11551050
100	Orange Julius	11552200
100	Fruit smoothie drink, made with fruit or fruit juice and dai	11553000
100	Fruit smoothie drink, NFS	11553100
100	Chocolate-flavored drink, whey- and milk-based	11560000
100	Flavored milk drink, whey- and milk-based, flavors other tha	11560020
100	Instant breakfast, powder, milk added	11612000
100	Instant breakfast, powder, sweetened with low calorie sweete	11613000
100	Cream, NS as to light, heavy, or half and half	12100100
100	Cream, light, fluid	12110100
100	Cream, light, whipped, unsweetened	12110300
100	Cream, half and half	12120100
100	Cream, half and half, fat free	12120110
100	Cream, heavy, fluid	12130100
100	Cream, heavy, whipped, sweetened	12140000
100	Sour cream	12310100
100	Sour cream, reduced fat	12310300
100	Sour cream, light	12310350
100	Sour cream, fat free	12310370
100	Dip, sour cream base	12350000
100	Dip, sour cream base, reduced calorie	12350020
100	Spinach dip, sour cream base	12350100
100	Ice cream, NFS	13110000
100	Ice cream, regular, flavors other than chocolate	13110100
100	Ice cream, regular, chocolate	13110110
100	Ice cream, rich, flavors other than chocolate	13110120
100	Ice cream, rich, chocolate	13110130
100	Ice cream, soft serve, flavors other than chocolate	13110200

% Milk in Food Item	Food Item Description	USDA Food Code
100	Ice cream, soft serve, chocolate	13110210
100	Ice cream, soft serve, NS as to flavor	13110220
100	ICE CREAM W/ SHERBET	13125100
100	Ice cream, fried	13126000
100	Light ice cream, flavors other than chocolate (formerly ice	13130300
100	Light ice cream, chocolate (formerly ice milk)	13130310
100	Light ice cream, no sugar added, NS as to flavor	13130320
100	Light ice cream, no sugar added, flavors other than chocolat	13130330
100	Light ice cream, no sugar added, chocolate	13130340
100	LIGHT ICE CREAM,PREMIUM, NOT CHOC (FORMERLY ICE MILK)	13130350
100	Light ice cream, soft serve, NS as to flavor (formerly ice m	13130590
100	Light ice cream, soft serve, flavors other than chocolate (f	13130600
100	Light ice cream, soft serve cone, chocolate (formerly ice mi	13130630
100	Light ice cream, soft serve cone, NS as to flavor (formerly	13130640
100	Light ice cream, cone, chocolate (formerly ice milk)	13140550
100	Light ice cream, sundae, soft serve, chocolate or fudge topp	13140660
100	Light ice cream, sundae, soft serve, not fruit or chocolate	13140680
100	LIGHT ICE CREAM,W/ SHERBET OR ICE CREAM (FORMERLY ICE MILK)	13141100
100	Sherbet, all flavors	13150000
100	MILK DESSERT, FROZEN, MADE FROM LOWFAT MILK	13160000
100	MILK DESSERT,FZN,LOWFAT,W/LOW CAL SWEET,NOT CHOC	13160100
100	Fat free ice cream, no sugar added, chocolate	13160150
100	Fat free ice cream, no sugar added, flavors other than choco	13160160
100	MILK DESSERT,FROZEN,LOWFAT,NOT CHOCOLATE	13160200
100	MILK DESSERT, FROZEN, LOWFAT, CHOCOLATE	13160210
100	Fat free ice cream, flavors other than chocolate	13160400
100	Fat free ice cream, chocolate	13160410
100	MILK DSRT,FROZ,MILK-FAT FREE,W/SIMPLESSE, NOT CHOC	13160550
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, NOT CHOC	13160600
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, CHOCOLATE	13160650
100	Milk dessert sandwich bar, frozen, made from lowfat milk	13161500
100	Milk dessert bar, frozen, made from lowfat milk and low calo	13161600
100	Light ice cream, bar or stick, with low-calorie sweetener, c	13161630

% Milk in Food Item	Food Item Description	USDA Food Code
100	Pudding, NFS	13200110
100	Pudding, chocolate, ready-to-eat, NS as to from dry mix or c	13210220
100	Pudding, chocolate, ready-to-eat, low calorie, containing ar	13210250
100	Pudding, flavors other than chocolate, ready-to-eat, NS as t	13210280
100	Pudding, flavors other than chocolate, ready-to-eat, low cal	13210290
100	Custard	13210300
100	Custard, Puerto Rican style (Flan)	13210350
100	Pudding, rice	13210410
100	Pudding, tapioca, made from home recipe, made with milk	13210500
100	Pudding, tapioca, made from dry mix, made with milk	13210520
100	Pudding, coconut	13210610
100	Puerto Rican pumpkin pudding (Flan de calabaza)	13210810
100	Pudding, flavors other than chocolate, prepared from dry mix	13220110
100	Pudding, chocolate, prepared from dry mix, milk added	13220120
100	Pudding, flavors other than chocolate, prepared from dry mix	13220210
100	Pudding, chocolate, prepared from dry mix, low calorie, cont	13220220
100	Mousse, chocolate	13250000
100	Milk dessert or milk candy, Puerto Rican style (Dulce de lec	13252200
100	Barfi or Burfi, Indian dessert, made from milk and/or cream	13252500
100	Tiramisu	13252600
100	Custard pudding, flavor other than chocolate, baby food, NS	13310000
100	Custard pudding, baby food, flavor other than chocolate, str	13311000
100	Custard pudding, baby food, flavor other than chocolate, jun	13312000
100	White sauce, milk sauce	13411000
100	Milk gravy, quick gravy	13412000
100	Cheese, NFS	14010000
100	Cheese, Cheddar or American type, NS as to natural or proces	14010100
100	Cheese, natural, NFS	14100100
100	Cheese, Blue or Roquefort	14101010
100	Cheese, Brick	14102010
100	Cheese, Brie	14103020
100	Cheese, natural, Cheddar or American type	14104010
100	Cheese, Cheddar or American type, dry, grated	14104020
100	Cheese, Colby	14104200

% Milk in Food Item	Food Item Description	USDA Food Code
100	Cheese, Colby Jack	14104250
100	Cheese, Feta	14104400
100	Cheese, Fontina	14104600
100	Cheese, goat	14104700
100	Cheese, Gouda or Edam	14105010
100	Cheese, Gruyere	14105200
100	Cheese, Monterey	14106200
100	Cheese, Monterey, lowfat	14106500
100	Cheese, Mozzarella, NFS	14107010
100	Cheese, Mozzarella, whole milk	14107020
100	Cheese, Mozzarella, part skim	14107030
100	Cheese, Mozzarella, nonfat or fat free	14107060
100	Cheese, Muenster	14107200
100	Cheese, Muenster, lowfat	14107250
100	Cheese, Parmesan, dry grated	14108010
100	Cheese, Parmesan, hard	14108020
100	Cheese, Parmesan, low sodium	14108050
100	Parmesan cheese topping, fat free	14108060
100	Cheese, Provolone	14108400
100	Cheese, Swiss	14109010
100	Cheese, Swiss, low sodium	14109020
100	Cheese, Swiss, lowfat	14109030
100	Cheese, Cheddar or Colby, low sodium	14110010
100	Cheese, Cheddar or Colby, lowfat	14110030
100	Cheese, Mexican blend	14120010
100	Queso Anejo (aged Mexican cheese)	14131000
100	Queso Asadero	14131500
100	Queso Chihuahua	14132000
100	Queso Fresco	14133000
100	Cheese, cottage, NFS	14200100
100	Cheese, cottage, creamed, large or small curd	14201010
100	Cottage cheese, farmer's	14201200
100	Cheese, Ricotta	14201500
100	Cheese, cottage, with fruit	14202010

% Milk in Food Item	Food Item Description	USDA Food Code
100	Cheese, cottage, salted, dry curd	14203020
100	Puerto Rican white cheese (queso del pais, blanco)	14203510
100	Cheese, cottage, lowfat (1-2% fat)	14204010
100	Cheese, cottage, lowfat, with fruit	14204020
100	CHEESE, YOGURT, NFS	14210000
100	Cheese, cream	14301010
100	Cheese, cream, light or lite (formerly called Cream Cheese L	14303010
100	Cheese spread, cream cheese, regular	14420200
100	Cheese, cottage cheese, with gelatin dessert	14610200
100	Topping from cheese pizza	14620300
100	Topping from vegetable pizza	14620310
100	Topping from meat pizza	14620320
100	Cheese fondue	14630100
100	Cheese sauce	14650100
100	Cheese sauce made with lowfat cheese	14650150
100	Alfredo sauce	14650160

Appendix E

Determination of Chemicals for Multipathway Analysis

E.1 Introduction

The AB-2588 program assesses the risk from airborne chemicals that are often emitted by facilities at high temperature and pressure in the presence of particulate matter. Some of these chemicals will be emitted and remain in vapor form. The inhalation cancer risk and noncancer hazard from such volatile chemicals are likely to be much greater than the risk from other possible exposure pathways. Other chemicals, such as semi-volatile organic or metal toxicants, can either be emitted as particles, form particles after emission from the facility, or adhere to existing particles. Some chemicals will partition between the vapor and particulate phases. Some chemicals such as PAHs have been found to have a portion of the particle associated mass in reversible equilibrium with the vapor phase and a portion irreversibly bound (Eiceman and Vandiver, 1983). Chemicals in the particulate phase can be removed from the atmosphere by settling. The settling of smaller particles can be enhanced by coalescence into larger particles with greater mass.

There are a number of exposure pathways by which humans may be exposed to airborne chemicals. Particulate associated chemicals can be deposited directly onto soil, onto the leaves of crops, or onto surface waters. Crops may also be contaminated by root uptake of chemicals. Livestock such as chickens, pigs and cows may be contaminated by inhalation of such chemicals or by consumption of contaminated feed, pasture, or surface waters. Humans may be exposed to these chemicals through inhalation, consumption of crops, soil, surface waters, meat, eggs and dairy products. Infants may be exposed through consumption of human breast milk.

E.2 Criteria for Selection of Chemicals for Multipathway Analysis

Chemicals listed in Appendix A, "Substances for Which Emissions Must be Quantified" which have been previously reported to be emitted by facilities in California under the Air Toxics "Hot Spots" Act were considered as candidates for multipathway analysis. From the chemicals meeting this criteria, chemicals which had been considered in the past to be multipathway chemicals or were thought to be likely candidates were selected for further analysis. We chose chemicals on the basis of whether they might be particle bound.

Junge (1977) developed a theoretical model for the partitioning of the exchangeable fraction of an airborne chemical between the vapor and particulate phases in the ambient air.

$$\theta = \frac{bS^{(p)}}{P_L^s + bS^{(p)}}$$

Where:

θ = fraction of the total mass of chemical on the particle phase
(unitless)

b = a constant (mm Hg cm³/cm²)

$S^{(p)}$ = total surface area of particle per unit volume of air (cm²/cm³)

P_L^s = saturation pressure of the liquid chemical at ambient temperature (mm Hg)

Junge (1977) did not distinguish between solid and liquid phase vapor pressures. Pankow (1987) recognized the importance of using the liquid phase vapor pressure. When the chemical of interest is a solid at the temperature of interest, the subcooled liquid vapor pressure must be used. The subcooled liquid vapor pressure is an extrapolation of the saturated liquid vapor pressure below the melting point where the compound actually exists as solid (Boethling and McKay, 2000). The subcooled liquid vapor pressure can be estimated using the following equation:

$$P_L^s/P_s^s = \exp[\Delta S_f (T_m - T)/RT]/RT$$

Where:

P_L^s = sub cooled liquid vapor pressure of the liquid chemical at ambient temperature (Pascal).

P_s^s = saturated vapor of the solid at room temperature

ΔS_f = entropy of fusion (J/mol K)

T_m = melting point temperature (K)

T = ambient temperature (K)

R = gas constant (8.3143 joules/K mole)

Values for ΔS_f may be obtained in the literature. In cases where a literature value is not available a default value of 56.45 has been suggested by Boethling and McKay (2000).

The percentage of the total mass of chemical (vapor plus particulate fractions) is determined by multiplying θ times 100. The percentage of the total mass of chemical that is in particulate phase is determined in part by the concentration of particles in the air. For our purposes, we used an average concentration of particles in urban air determined by Whitby, (1978). The concentration of particles was 1.04×10^{-4} $\mu\text{g}/\text{cm}^3$. The surface area per μg of particle was assumed to be $0.05 \text{ cm}^2/\mu\text{g}$. Thus the $S^{(p)}$ is calculated to be 5.2×10^{-6}

cm^2/cm^3 . The value of b used is the default value of $0.1292 \text{ mm Hg cm}^3/\text{cm}^2$ recommended by Pankow (1987).

It should be noted that the particle bound associated fraction of some semi-volatile organic toxicants has been found to consist of a non-exchangeable fraction and a fraction which equilibrates with the vapor phase (Bidleman and Foreman, 1987). The equation of Junge (1977) only addresses the exchangeable fraction. This means that the actual fraction of the total mass that is particle bound material may be somewhat higher than the theoretical model which Junge (1977) proposed. The partitioning of semi-volatile organic toxicants between the vapor phase and particles has been experimentally investigated by Bidleman et al., (1986) and Bidleman and Foreman (1987). High volume sampling has been done in several cities in which the particulate and vapor fractions have been collected on filters and adsorbents. This work has supported the validity of the theoretical model of Junge (1977).

The Junge (1977) and Pankow (1987) model appears to be the best available to develop criteria to determine which chemicals emitted by facilities in the AB-2588 program should undergo multipathway analysis. The liquid or subcooled liquid vapor pressure at ambient temperatures determines the fraction of chemical that will be particle associated. The vapor pressure is available for most of the chemicals for which the determination needs to be made.

It should be noted that the Junge (1977) model was designed to look at the partitioning of chemicals between the particle and vapor phases under equilibrium conditions in the atmosphere. The initial conditions under which particle formation may occur as chemicals are emitted into the atmosphere may be different from the conditions assumed by Junge (1977). The chemicals of concern in the AB-2588 program may be emitted at high temperatures and pressures in the presence of a high concentration of particulate matter. Such conditions may favor partitioning of mass toward the particulate fraction. It is also possible that such conditions might favor the formation of a greater fraction of non-exchangeable particle associated chemical which is not taken into account in the Junge (1977) equation. The rapid cooling from high temperature to ambient temperature may also influence the percent of total mass which is particle bound in ways that are not accounted for in the simple equilibrium model of Junge (1977).

OEHHA has decided that chemicals with less than 0.5% of the total mass in the particle-associated fraction will not be considered for multipathway analysis. The 0.5% is a relatively small percentage of the total mass. This percentage was chosen in part to compensate for the uncertainties involved in extrapolation of the Junge (1977) model to the conditions under which particles may be formed in the stacks of facilities. Thus chemicals with vapor pressures greater than $1.34 \times 10^{-4} \text{ mm Hg}$ at 25°C will not be considered for multipathway analysis. An exception to this rule is the inclusion of hexachlorobenzene (HCB) for

multipathway analysis, even though its calculated percentage of total mass in the particulate phase is expected to be below 0.5%. The criteria for including HCB is discussed in Section E.3 below. It should be noted that the chemicals for which noninhalation pathway risks are a significant fraction of the total risk are metals, PAH's, PCB's, polychlorinated dibenzo-p-dioxins and furans. These chemicals have much higher percentages of total mass in the particulate fraction than 0.5%.

There are some toxic compounds without measurable vapor pressure at 25°C such as the metals and their compounds. These metals include lead, mercury compounds, nickel, selenium, fluoride, beryllium, arsenic, chromium VI and cadmium. These toxicants are included on the list of chemicals for multipathway analysis.

In Table E.1 we have calculated the air/particle partition coefficients of the compounds emitted by facilities for which it appeared possible that a significant fraction of the total mass could be in the particulate fraction. In some cases the saturated vapor pressure at a temperature at or near ambient temperature (25°C) the air/particle coefficient could not be calculated.

For PAHs, consideration for multipathway analysis is largely confined to PAHs with 4 or more fused rings because a significant fraction of their total mass is in the particle phase. Naphthalene contains 2 fused rings and is included in the Hot Spots program as a carcinogen. However, it does not have a significant percentage of its total mass in the particle phase, so is not considered for multipathway analysis. The PAHs with 3 fused rings (e.g., phenanthrene, fluorine, acenaphthene) are also predominantly found in gaseous form and the data is currently too limited or inadequate to list any of them as carcinogens. Laboratory studies of sludge-amended soils containing PAHs have also shown significant loss through volatilization only for PAHs with less than 4 fused rings (Wild and Jones, 1993). Thus, speciated analysis for PAHs that include only the compounds with 4 or more fused rings can be used for multipathway assessment.

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
4,4'methylene dianiline	1.0	197	1	NA	NA
o-cresol	0.28*	38.2,	2	2.44×10^{-6}	2.44×10^{-4}
m-cresol	0.39**	25	2	1.71×10^{-6}	1.71×10^{-4}
p-cresol	0.37**	25	2	1.81×10^{-6}	1.81×10^{-4}
Cellosolve	5.63***	25	3	1.19×10^{-7}	1.19×10^{-5}
Cellosolve acetate	2.12***	25	3	3.17×10^{-7}	3.19E-05
Mercury (elemental)	1.20×10^{-3} ***	25	4	5.6×10^{-4}	0.056
Hexachlorocyclohexanes (Lindane)	1.18×10^{-4} **	20	5	5.66×10^{-3}	0.57
Phthalates					
Diethylhexylphthalate	1.97×10^{-7} ***	25	3	7.73×10^{-1}	77.3
Chlorobenzenes					
Chlorobenzene	12.2***	25	6	5.53×10^{-8}	5.53×10^{-6}
p-Dichlorobenzene	0.65***	25	6	1.03×10^{-6}	9.93×10^{-5}
m-Dichlorobenzene	2.30***	25	6	1.03×10^{-6}	1.03×10^{-4}
o-Dichlorobenzene	0.39***	25	6	1.71×10^{-6}	1.71×10^{-4}
1,2,3-Trichlorobenzene	0.39*	40	6	1.71×10^{-6}	1.71×10^{-4}
1,2,4-Trichlorobenzene	0.45*	38	6	1.48×10^{-6}	1.48×10^{-6}
1,2,3,4-Tetrachlorobenzene	6.58×10^{-7}		6	1.02×10^{-5}	1.02×10^{-3}

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
	^{2*}				
1,2,3,5-Tetrachlorobenzene	0.14*		6	4.82×10^{-6}	4.82×10^{-4}
Pentachlorobenzene	$6.67 \times 10^{-3*}$	25	6	1.01×10^{-4}	1.01×10^{-2}
Hexachlorobenzene	$2.96 \times 10^{-4*}$	25	6	2.96×10^{-4}	2.96×10^{-2}

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals (Cont.).

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
PAHs					
Naphthalene (2 fused rings)	0.31*	25	7	2.14×10^{-6}	2.14×10^{-4}
Acenaphthene (3 fused rings)	$3.02 \times 10^{-3*}$	25	7	2.23×10^{-5}	2.23×10^{-3}
Acenaphthylene (3 fused rings)	6.67×10^{-3}	25	7	1.00×10^{-4}	0.01
Anthracene (3 fused rings)	$4.2 \times 10^{-6*}$	25	7	1.57×10^{-2}	1.57
Benzo[a]anthracene (4 fused rings)	$4.07 \times 10^{-6*}$	25	7	1.42×10^{-1}	14.2
Chrysene (4 fused rings)	$8.81 \times 10^{-8**}$	25	7	8.84×10^{-1}	88.4
Benzo[a]pyrene (5 fused rings)	9.23×10^{-8}	25	7	8.79×10^{-1}	87.9
Benzo[b]fluoranthene (5 fused rings)	1.59×10^{-7}	25	7	8.09×10^{-1}	80.9
Benzo[k]fluoranthene (5 fused rings)	$3.7 \times 10^{-8*}$	25	7	9.48×10^{-1}	94.8
Dibenz[a,h]anthracene (5 fused rings)	$6.07 \times 10^{-11**}$	25	7	1.00×10^0	100
Indeno[1,2,3-cd]pyrene (6 fused rings)	$1.19 \times 10^{-9**}$	25	8	9.98×10^{-1}	99.8
Chlorophenols					
Pentachlorophenol	$1.73 \times 10^{-3*}$	25	2	3.88×10^{-4}	3.88×10^{-2}
2,4,6-Trichlorophenol	$2.8 \times 10^{-02*}$	25	2	2.34×10^{-5}	2.34×10^{-3}
2,4,5-Trichlorophenol	$4.59 \times 10^{-02*}$	25	2	1.46×10^{-5}	1.46×10^{-3}
Nitrosoamines					
N-Nitrosodiethylamine	8.60×10^{-7}	20	1	7.81×10^{-7}	7.81×10^{-5}

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
	^{1***}				
N-Nitrosodimethylamine	8.1***	20	2	8.29×10^{-8}	8.29×10^{-6}
N-Nitrosodiphenylamine	$4.12 \times 10^{-2**}$	25	2	1.63×10^{-9}	1.63×10^{-7}
N-Nitrosodi-n-butylamine	$3.00 \times 10^{-2***}$	20	9	2.24×10^{-5}	2.24×10^{-3}
N-Nitrosodi-n-propylamine	$4.15 \times 10^{-1***}$	20	2	1.62×10^{-6}	1.62×10^{-4}
N-Nitrosopyrrolidine	$7.20 \times 10^{-02***}$	20	9	9.2×10^{-6}	9.2×10^{-4}

Table E1 **Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals (Cont.).**

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
PCBs					
Aroclor 1016	$1.50 \times 10^{-3*}$	25	6	4.48×10^{-4}	4.48×10^{-2}
Aroclor 1221	$1.50 \times 10^{-2*}$	25	6	4.48×10^{-5}	4.48×10^{-3}
Aroclor 1232	$4.05 \times 10^{-03***}$	25	6	1.66×10^{-4}	0.17
Aroclor 1242	$4.13 \times 10^{-04***}$	25	6	1.63×10^{-4}	0.16
Aroclor 1248	$3.33 \times 10^{-04***}$	25	6	1.66×10^{-3}	0.17
Aroclor 1254	$7.73 \times 10^{-05***}$	25	6	8.62×10^{-3}	0.86
Aroclor 1260	$4.40 \times 10^{-06***}$	25	6	1.32×10^{-1}	13.2
Dioxins and Furans					
2,3,7,8 Tetrachlorodibenzo-p-dioxin	$4.5 \times 10^{-7*}$	20	7	5.97×10^{-1}	59.7
2,3,7,8 Tetrachlorodibenzofuran	$9.21 \times 10^{-7*}$	25	7	9.97×10^{-1}	99.7
1,2,3,4,7 Pentachlorodibenzodioxin	$5.9 \times 10^{-7**}$	25	7	5.42×10^{-1}	54.2
2,3,4,7,8 Pentachlorodibenzofuran	$1.63 \times 10^{-7*}$	25	7	4.22×10^{-1}	42.2
1,2,3,4,7,8 Hexachlorodibenzo-p-dioxin	$5.89 \times 10^{-9*}$	25	7	9.17×10^{-1}	91.7
1,2,3,4,7,8 Hexachlorodibenzofuran	$6.07 \times 10^{-8*}$	25	7	9.89×10^{-1}	98.9
1,2,3,4,6,7,8 Heptachlorodibenzo-p-dioxin	$7.68 \times 10^{-9*}$	25	7	9.76×10^{-1}	97.6
1,2,3,4,6,7,8 Heptachlorodibenzofuran	$1.68 \times 10^{-8*}$	25	7	9.76×10^{-1}	97.6
1,2,3,4,7,8,9 Heptachlorodibenzofuran	$9.79 \times 10^{-9*}$	25	7	9.87×10^{-1}	98.7

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particulate (of total mass)
1,2,3,4,5,6,7,8 Octachlorodibenzofuran	$1.95 \times 10^{-9*}$	25	7	9.97×10^{-1}	99.7
1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin	$2.08 \times 10^{-9*}$	25	7	9.97×10^{-1}	99.7

1. IARC, 1986; 2. McKay et al. 1992a; 3. McKone et al., 1993; 4. Cohen et al., 1994; 5. ATSDR, ???; 6. McKay et al., 1992b; 7. McKay et al., 1992c; 8. Montgomery and Welkom, 1990; 9. Klein, 1982

*Indicates subcooled liquid vapor pressure

Indicates subcooled liquid vapor pressure estimated according to Boethling and McKay, 2000, page 238. *Indicates Psat liquid (substance is a liquid at 25 °C)

For the nitrosamines, we were not able to locate saturated vapor pressures for N-nitrosomethylethylamine, N-nitrosomorpholine, and N-nitrosopiperidine. We were able to find saturated vapor pressures for N-nitrosodiethylamine, N-nitrosodimethylamine, N-nitrosodiphenylamine, N-nitrosodi-n-butylamine, N-nitrosodi-n-propylamine and N-nitrosopyrrolidine. None of these compounds had particle associated percentages above 0.5%. N-nitrosopyrrolidine was structurally similar to N-nitrosomorpholine and N-nitrosopiperidine. N-nitrosopyrrolidine has a particle associated percentage of 9.2×10^{-4} . This is well below the 0.5% that we selected as our cutoff. We therefore felt that N-nitrosomorpholine and N-nitrosopiperidine were unlikely to have a particle bound percentage above 0.5% and thus we excluded these compounds from multipathway consideration. N-nitrosomethylethylamine did not appear likely to have a particle bound percentage above N-nitrosodiethylamine, N-nitrosodimethylamine or N-nitrosodi-n-butylamine. All of these nitrosamines are well below the 0.5% cutoff.

Table E2. Chemicals for Which Multipathway Risks Need to be assessed.

4,4'-methylene dianiline¹
creosotes
diethylhexylphthalate
hexachlorobenzene
hexachlorocyclohexanes
PAHs (including but not limited to the following:)²
 benz[a]anthracene
 benzo[b]fluoranthene
 benzo[j]fluoranthene
 benzo[k]fluoranthene
 benzo[a]pyrene
 dibenz[a,h]acridine
 dibenz[a,j]acridine
 7H-dibenzo[c,g]carbazole
 7,12-dimethylbenz[a]anthracene
 3-methylcholanthrene
 5-methylchrysene
 dibenz[a,h]anthracene
 dibenzo[a,e]pyrene
 dibenzo[a,h]pyrene
 dibenzo[a,i]pyrene
 dibenzo[a,l]pyrene
 chrysene
 indeno[1,2,3-cd]pyrene

PCBs³

Polychlorinated dibenzo-p-dioxins {PCDDs} (including but not limited to the following, but excluding dioxins with less than four chlorines:)⁴

2,3,7,8 tetrachlorodibenzo-p-dioxin
1,2,3,7,8 pentachloro-p-dioxin
1,2,3,4,7,8 hexachlorodibenzo-p-dioxin
1,2,3,6,7,8 hexachlorodibenzo-p-dioxin

Table E2. Chemicals for Which Multipathway Risks Need to be Assessed (Cont.).

1,2,3,7,8,9 hexachlorodibenzo-p-dioxin
1,2,3,4,6,7,8 heptachlorodibenzo-p-dioxin
1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin

Polychlorinated dibenzofurans {PCDFs} (including but not limited to the following, but excluding dibenzofurans with less than four chlorines:)⁴

- 2,3,7,8 tetrachlorodibenzofuran
- 1,2,3,7,8 pentachlorodibenzofuran
- 2,3,4,7,8 pentachlorodibenzofuran
- 1,2,3,4,7,8 hexachlorodibenzofuran
- 1,2,3,6,7,8 hexachlorodibenzofuran
- 1,2,3,7,8,9 hexachlorodibenzofuran
- 2,3,4,6,7,8 hexachlorodibenzofuran
- 1,2,3,4,6,7,8 heptachlorodibenzofuran
- 1,2,3,4,7,8,9 heptachlorodibenzofuran
- 1,2,3,4,5,6,7,8 Octachlorodibenzofuran

arsenic and arsenic compounds
beryllium and beryllium compounds
cadmium and cadmium compounds
soluble compounds of chromium VI
fluoride and soluble fluoride compounds
lead and inorganic lead compounds
inorganic mercury compounds
nickel and nickel compounds
selenium and selenium compounds

¹ The saturated vapor pressure at 25°C or close to 25°C, is not available to our knowledge. The other evidence available, a melting point of 91.5°C and a boiling point of 398-399 °C (Merck, 1989) indicate that it is very likely that a very significant fraction of the chemical emitted into the air would be in the particulate phase. In addition the vapor pressure at 197 °C is only 1 mm (IARC, 1986).

² PAHs with four or more fused rings (Table E2) are to be assessed for multipathway analysis. If PAH mixtures are not reported, rather than as specific PAHs, then the cancer potency of the entire mixture should be treated the same as benzo(a)pyrene / 10. If the proportion of a PAH mixture greater than 3 fused rings are known in the mixture but not speciated, then the cancer potency should be treated the same as benzo(a)pyrene.

³ PCBs is inclusive of all Aroclor mixtures. The information in Table E1 indicates that some of the Aroclor mixtures do not have significant air/particle coefficients. However, it is difficult to determine vapor pressures on mixtures of compounds. OEHHHA therefore is proposing to include all of the Aroclors in the list of chemicals for multipathway analysis. The percentage of some individual PCBs in the particulate phase has been measured in air samples (Horstmann and McLachlan, 1998). The particulate phase of tetrachlorinated PCBs (PCB 152) can be expected to be around 1.4%, and increasing to 11.3% for the heptachlorinated PCBs (PCB 180)

⁴ From OEHHA analysis (Table E1), it is clear that all polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans should be included in the multipathway analysis.

Table E3 Specific Pathways to be Analyzed for Multipathway Chemicals

Chemical	Soil Ingestion	Dermal	Meat, Milk & Egg	Fish Ingestion	Expos Veg. Ingest.	Leafy Veg. Ingest.	Protecte d. Veg	Root Veg. Ingest.	Water Ingest	Breast Milk Ingestion
4,4'-methylene dianiline	X	X		X	X	X			X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X	X	X	X	X			X	
Hexachlorocyclohexanes	X	X	X	X	X	X			X	
Hexachlorobenzene	X	X	X	X	X	X			X	
PAHs	X	X	X	X	X	X			X	X
PCBs	X	X	X	X	X	X			X	X
Dioxins & furans	X	X	X	X	X	X			X	X
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & compounds	X	X	X	X	X	X	X	X	X	
Inorganic arsenic & cmpds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	X
Inorganic mercury cmpds	X	X	X	X	X	X	X	X	X	
Nickel & compounds	X	X	X	X	X	X	X	X	X	
Fluoride & compounds	X	X	X	X	X	X	X	X	X	
Selenium and compounds	X	X	X	X	X	X	X	X	X	

OEHHA is recommending that all of the chemicals chosen for multipathway analysis be included in the soil ingestion and dermal pathways. The soil t1/2 values needed to determine concentration in the soil are found in Appendix G. The variates need for the dermal pathway are found in Chapter 6 and Appendix F.

The meat (beef, chicken, pork), cow's milk and egg pathways are listed in one column because the lipid solubility and half life in the body are common factors which determine if these compounds will be present in these three pathways in appreciable concentrations in the fat of meat, milk and eggs.

E.3 Evidence for Inclusion of Hexachlorobenzene for Multipathway Assessment

In the previous Hot Spots Guidance document, semi-volatile substances with less than 0.5% of their total mass in the particle-associated fraction was not considered for multipathway analysis. Although this is a reasonable cut-off for semi-volatile substances predominantly in the gas phase, an exception is made for hexachlorobenzene (HCB). From Table E1, the Junge model shows HCB with a particle/gas ratio of only 0.0296% at 25 °C. Normally, this would exclude HCB from multipathway analysis. However, actual field measurements of the air/particle partitioning of HCB in Table E.4 shows that the compound is often found in particle form above 0.5%.

The greater than expected particle fraction for HCB is a likely result of environmental conditions at the locations assessed for HCB. The adsorption of HCB on aerosols and subsequent deposition depends on the vapor pressure, the amount and surface area of aerosol particles, and the relevant environmental temperature (Ballschmiter & Wittlinger, 1991). Colder temperatures and greater airborne particulate levels would increase the particle/gas ratio of HCB. In fact, Ballschmiter & Wittlinger (1991) suggested that the particle fraction found at -8 °C (3.5%) in a rural region will be similar to the particle fraction in urban areas with higher particulate levels and an air temperature of 15 °C.

Table E.4. Field study vapor/particle distributions of HCB

Study	Particle fraction Concentration (% particle)	Gas phase Concentration (% gas)
Popp et al., 2000 ^a		
Leipzig area	0.8 pg/Nm ³ (0.9%)	83.1 pg/Nm ³ (99.1%)
Roitzsch area	0.5 pg/Nm ³ (0.3%)	145.6 pg/Nm ³ (99.7%)
Greppin area	2.6 pg/Nm ³ (0.9%)	280.6 pg/Nm ³ (99.1%)
Horstmann & McLachlan, (1998) ^b	0.43 pg/m ³ (0.2%)	210 pg/m ³ (99.8%)
Lane et al., 1992 ^c		
Turkey lake	3 pg/m ³ (4.1%)	71 pg/m ³ (95.9%)
Pt. Petre	2 pg/m ³ (2.8%)	69 pg/m ³ (97.2%)
Ballschmiter & Wittlinger, 1991 ^d	4 pg/m ³ (3.5%)	110 pg/m ³ (96.5%)
Bidleman et al., 1987 ^e		
20 °C	(nd) ^f (0.1%)	(nd) (99.9%)
0 °C	(nd) (0.7%)	(nd) (99.3%)

^a Air samples collected near chlorobenzene-contaminated sites of Bitterfeld region in Germany over a two-week period during the summer of 1998.

^b Air samples collected over one year in a forest clearing in Germany from May 1995 to April 1996.

^c Air samples collected during spring, summer, and fall of 1987 in rural regions of Ontario, Canada.

^d Air sample taken at a mean ambient temperature of -8 °C outside a small village near a major road in Germany

^e Data collected from Stockholm, Denver and Columbia. Vapor phase component possibly overestimated due to volatilization (blowoff) from the particle phase in the sampler.

^f No concentration data was provided.

In addition, Foreman and Bidleman (1987) have suggested that field measurements of HCB particle fractions may be greater than in laboratory settings because sources in the environment includes combustion-derived HCB particle incorporation. Similar to dioxins, combustion of organic material that includes chlorinated substances has been suggested as a primary source of HCB.

Nevertheless, the minor particle fraction of the HCB results in Table E.4 may still not be sufficient to support a multipathway analysis. However, when the extreme environmental persistence of this compound relative to other predominantly gaseous semi-volatile substances (i.e., nitrosamines and chlorophenols) is taken into account, it appears that even a fraction of the compound depositing in the particle bound phase could result in measurable levels in sediment and soil with possible accumulation over time. Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from atmospheric sources, have found that HCB accumulated in water, sediment and fish tissue

samples (Eisenreich et al., 1981). In particular, the strong retention of HCB to sediment particulates in the water allowed much of the historical burden to become immobilized in bottom sediments, with a concomitant reduction in the levels of HCB found in the surface waters.

More evidence for HCB's persistence in soil was observed in a laboratory study. Aerial application of HCB in a greenhouse with simulated pasture conditions showed that HCB volatilized fairly rapidly from plant and soil surfaces (Beall, 1976). Only 3.4% of HCB remained in the top 2 cm of soil 19 months after spraying. Residues on the grass grown in the soil volatilized considerably faster, with only 1.5% remaining on the plants after two weeks, and <0.01% at 19 months. However, no significant reduction in HCB was found in the deeper 2-4 cm layer of soil after 19 months, showing HCB to be persistent within the soil, including a resistance to microbial degradation and leaching. The immobilization of HCB within the soil is due to its high K_{ow} , leading to strong adsorption to the soil organic fraction.

E.4 *Summary*

The theoretical model of Junge (1977) uses the liquid or subcooled liquid vapor pressure to determine the percentage of the total airborne mass of chemical that is particulate. Chemicals with 0.5% of the total mass or more in the particulate fraction at 25° C are considered by OEHHHA to be multipathway chemicals. This corresponds to toxicants with a vapor pressure of 1.34×10^{-6} mm Hg. A list of multipathway chemicals for the AB-2588 program is provided in Table E2. The percentage of the total mass in the particulate phase and the air/particle partition coefficients for these chemicals and a few other selected chemicals are presented in Table E1.

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Appendix F
Dermal Exposure to Soil-Bound Hot Spots Multipathway Chemicals:
Fractional Absorption (ABS) Values

F.1 Introduction

The absorbed dose resulting from dermal exposure to soil-bound chemicals depends on many factors. An algorithm that describes the uptake of chemicals from soil as a function of exposure duration, exposure frequency, chemical concentration in the soil, soil loading, surface area, body weight, averaging time, and fractional absorption (ABS) is discussed in Chapter 6. The purpose of this appendix is to summarize the derivation of the ABS for the “Hot Spots” multipathway chemicals and present the information used in the development of each chemical ABS. A general discussion of the diverse factors influencing dermal absorption of soil-bound chemicals is presented below preceding the chemical ABS summaries.

A small subset of organic and metallic compounds evaluated under the Hot Spots program is subject to deposition onto soil, plants and water bodies. Therefore, exposure can occur by pathways other than inhalation. These chemicals are semi-volatile or nonvolatile, and are therefore partially or wholly in the solid or liquid phase after being emitted. Fate and transport of the deposited chemical must then be estimated in order to assess the impact on soil, water and foods that humans come in contact with. The basis for the selection of these compounds as “Hot Spots” multipathway substances can be found in Appendix E. The organic compounds of relevance listed under the “Hot Spots” program include 4,4'-methylene dianiline, hexachlorocyclohexanes, di(2-ethylhexyl)phthalate (DEHP), polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs). The metal or metalloid compounds of relevance include the inorganic salts of arsenic, beryllium, cadmium, fluoride, mercury, lead, nickel, selenium and hexavalent chromium.

F.1.1 Point Estimate Approach for ABS Derivation

An ABS is a chemical-dependent, scenario-dependent value that can vary with the characteristics of the soil matrix and the exposed population. Such characteristics include the relative lipophilicity/hydrophilicity of the compound, soil organic content, soil particle size, soil aging of the chemical, residence time on the skin, and exposed surface area. Some of these issues are discussed in greater detail in Chapter 6. The data necessary to characterize the variability in these variates are often not available. For this reason, the ABS values derived in this document are point estimates. In particular, site specific information on soil organic content and soil particle range are not available. These factors can have a significant impact on chemical absorption from soil and the uncertainty in the dose estimate from dermal absorption because of these and other factors can be large.

To derive a point estimate for a chemical, typically the value from the best and sometimes only study available was selected. If multiple studies were available

with data collected under similar conditions, the most comprehensive study was selected. Or if the studies were of equal reliability, their absorption values would be averaged for ABS determination. In some cases experimental data are not even sufficient for a point estimate ABS and a default ABS is recommended (see below).

F.1.2 Skin Morphology and Dermal Absorption Issues for ABS Determination

The transepidermal uptake of chemicals across skin involves a complex process of transport from the soil matrix to the external protective skin layer called the epidermis, and then through the epidermis to the underlying dermis. The outermost layer of the epidermis is called the stratum corneum, which is thought to provide the major barrier to the absorption of most substances deposited onto the skin surface. The stratum corneum in humans varies in thickness from about 5 μm to over 400 μm on the palms and soles of the feet (Poet and McDougal, 2002; Hostynek, 2003). Below lies the viable epidermis, about 50-100 μm thick, containing keratinocytes that proliferate and differentiate while moving upwards and replacing the stratum corneum cells as they wear away. Below the epidermis lies the hydrous tissue of the dermis perfused by the blood and lymphatic circulation.

Skin appendages, including hair follicles and sweat ducts, transit through all these layers and may provide an alternate pathway for dermal diffusion of some ions such as metal salts (Tregear, 1966; Flynn, 1990). However, skin appendages occupy only a fraction of the surface area of the skin, which may limit their potential as a major diffusion pathway into the systemic circulation.

During the transport through the viable-epidermal and dermal layers, metabolism may also play a role in the absorption process (Kao and Carver, 1990). Metabolism in the dermal layers could also activate a toxicant, resulting in skin as a target organ or producing toxicity elsewhere following systemic absorption. As noted above, specific dermal ABSs for soil-bound chemicals are difficult to obtain due in part to the complex multiphasic nature of the system and lack of published absorption data. Hawley (1985) suggested a default factor of 15 percent to correct for the effect of the soil matrix on the dermal uptake of organic chemicals. Experimental evidence, however, suggests absorption from soil will be chemical dependent. Hence, it is important to determine dermal uptake point estimate values for specific soil-bound chemicals where appropriate data are available, as they will be more accurate than those derived on broad-based assumptions.

To obtain the ABS, a measured amount of chemical in a given amount of soil is administered to the skin surface; this amount (wt chemical/area skin) is referred to as the applied dose. The amount of chemical that crosses the skin barrier is measured and the ABS is calculated by dividing the amount absorbed by the amount applied. When measurements are made in excreta or specific organs,

corrections are included for incomplete recovery. In experiments of this type, the administered amount (in soil or solvent) represents a finite level of application. The ABS so calculated is an experimental value that is dependent upon exposure conditions, such as length of exposure and extent of soil loading. The length of exposure used for dermal exposure assessment in this document is 24 hrs. A 24 hr exposure time is commonly used in dermal absorption studies, so it's compatible for ABS calculation. In instances where absorption data did not use 24 hr exposure, an ABS will generally be based on data that are near a 24 hr exposure.

In contrast to the studies that utilize the application of finite amounts of chemicals, dermal studies that mimic scenarios such as bathing and swimming, require the applications of infinite volumes, i.e. the volume of the administered dose is much larger than the volume of the exposed skin area and the chemical at the skin surface is continuously replenished. The latter exposure scenario is not applicable to the soil studies described in this chapter, although information obtained from such studies may be useful for discussion purposes. For additional information on dermal uptake of chemicals from water (or vapor), the reader is referred to U.S. EPA (2004). The dermal absorption of chemicals from dermal exposure to contaminated water is not addressed in the "Hot Spots" program because it is likely to be a minor contribution to overall dose if it occurs at all.

F.2 Risk Assessment Issues

Although all dermal absorption studies are useful for understanding the relationship between dermal exposure and absorption, the application of these studies to risk assessment involves specific issues that must be considered to avoid development of a point estimate that may greatly underestimate, or overestimate, the potential for dermal absorption. Included among these issues are biological characteristics, soil properties, and exposure scenarios, and the variability in each can introduce uncertainties into the point estimate determination of ABS. By understanding these issues, the implications of using experimentally derived dermal ABS can be better understood. Specific categories of issues that must be considered when assessing dermal absorption are discussed below.

F.2.1 Definition of dermal uptake

Comprehensive dermal absorption studies often include a quantitative analysis of the amount of chemical that has passed through skin into the systemic circulation (for in vivo studies) or appears in the receptor fluid (for in vitro studies), plus the amount of chemical remaining in the skin at the site of application. Fundamentally, dermal uptake/absorption refers to the amount of dermally applied chemical that is ultimately determined to be systemically available. Because absorbed chemicals may be retained in the skin for long periods of time

and act as a reservoir for the slow systemic absorption of chemicals, the chemical remaining in skin at the end of dermal absorption experiments is considered available for systemic absorption unless data are available that shows otherwise.

Some fraction of dermally-absorbed chemicals may be only superficially diffused into skin and deposit in the stratum corneum where they are subject to counter-current forces of skin shedding, or desquamation, and ultimately removed from the body before becoming systemically absorbed. Continuous desquamation with total stratum corneum turnover has been estimated to take 2-3 weeks (Hostynek, 2003). Modeling calculations by Reddy et al. (2000) indicate that epidermal turnover can significantly reduce subsequent chemical absorption into the systemic circulation for highly lipophilic ($\log K_{ow} > \text{about } 4$) or high molecular weight chemicals ($MW > \text{about } 350\text{-}400 \text{ Da}$). However, some highly lipophilic chemicals retained in skin at the end of dermal absorption studies have been shown to be predominantly available for eventual absorption into the systemic circulation. Chemicals of concern that fall into this category include the PAHs and DEHP (Chu et al., 1996).

Loss of absorbed chemical through skin shedding appears to occur more readily with some hydrophilic metal salts in which a portion of the metal becomes irreversibly bound in the epidermis and subject to eventual shedding with skin. Some metal salts have such a slow diffusion (i.e., long lag time) through skin that the stratum corneum turnover rate exceeds the chemical diffusion rate (Hostynek, 2003).

Tape stripping methods to remove thin layers of stratum corneum have been used in several studies discussed below to estimate the fraction of chemical in the stratum corneum that may be lost through desquamation. A more definitive approach used in a few cases is to extend the dermal uptake study for an additional few days (after chemical is removed from skin) to determine if more of the chemical retained in the skin becomes available for systemic absorption. Other studies that help determine the fate of chemicals retained in skin include skin localization techniques and skin binding studies (Miselnicky et al., 1988; Yourick et al., 2004). But in many instances the dermal uptake studies for individual chemicals did not provide enough data to determine the fate or location of the chemical retained in skin. Thus, as discussed above, the ABS will then represent that fraction of chemical still retained in skin, plus the fraction that has already passed through the skin.

F.2.2 Dermal Bioavailability of Chemicals in Soil

The term dermal bioavailability as it applies in this section refers to the fraction of chemical in soil that is actually dermally absorbed. Dermal bioaccessibility is another term used in reference to chemical-laden soils and represents that fraction of chemical solubilized from soil, usually into water, sweat, or

gastrointestinal fluids that then becomes available for absorption. By definition, bioaccessibility should exceed bioavailability.

Published data for some chemicals considered in this section contain only data for neat application of the chemical to skin in solvent or aqueous vehicle. Generally, there is a lack of absorption data for chemicals bound to soil. To avoid potential overestimation of absorption in these instances, bioaccessibility and soil leaching studies of soil-bound chemicals are considered for adjusting the fractional absorption of the pure chemical applied to skin. These studies can be used to determine the extractable, or bioaccessible, fraction of a soil pollutant that can be deposited on the skin surface. Water added to soil is often used to determine the bioaccessibility of a soil-bound chemical, although human sweat or synthetic sweat has also been used to estimate the amount of a pollutant that can be leached from contaminated soils (Horowitz and Finley, 1993; Filon et al., 2006; Nico et al., 2006).

F.2.3 Soil - Chemical - Tissue Interaction.

Soil is a complex matrix with a highly variable composition and absorptive capacity. Organic content, mineral composition, particle size, and pH are all highly variable. Because the dermal absorption of a compound from soil is often dependent on these characteristics, it follows that transfer of a chemical from soil particles to the skin surface for absorption is likely to vary with soil type.

Transfer of a chemical from soil particles to the skin surface is limited by the chemical's diffusion rate (McKone, 1990). Diffusion through the soil phase, through the air, and through soil moisture is all possible. Fugacity-based interphase transport models were constructed to describe the rate of each of these processes for chemicals in soil particles and to predict the dermal uptake rates. It was shown that predicted dermal uptake of chemicals from soil depends on the Henry's constant (vapor pressure/solubility in water), the octanol/water partition coefficient of a chemical, and the soil thickness on skin. If the Henry's constant is very high, chemicals will be lost from soil particles (or the skin surface) quite rapidly, so net dermal uptake of chemicals added to soil will be low. If the Henry's constant is very low, diffusion through the soil particle layer will be too slow to allow much dermal uptake unless the soil particles are very small. A high octanol/water partition coefficient is associated with tight binding to soil and low water solubility; these properties also limit the ability of a chemical to diffuse through the mixed lipid/water phases of the stratum corneum.

Other mathematical models have been developed by Bunge and Parks (1997) to describe dermal absorption of organic chemicals provided the chemical fits certain assumptions, such as falling within a defined octanol/water partition coefficient range ($1.59 \leq \log_{10}K_{ow} \leq 5.53$), and that the molecular weight of the organic chemical is ≤ 700 . Soil constraints for the model include contaminated soils with about 0.2% organic carbon or more, and with a clay fraction less than

60 times the weight fraction of organic carbon. The models were then used to estimate the relative effect of changing exposure conditions (e.g., changes in soil loading, contamination levels, chemical, etc.) compared to published experimental studies. Although the models were generally consistent with the experimental results for some chemicals, such as benzo(a)pyrene (BaP), they were considerably divergent from the experimental results for other chemicals, such as lindane (gamma-hexachlorocyclohexane).

The authors suggested that the fast soil release kinetics on which the models are based may not fit with what was observed experimentally for some chemicals (Bunge and Parks, 1997). Fast soil release kinetics assumes the primary resistance that controls transfer of the chemical from soil to skin resides in the dermal barrier, and that the kinetics of soil desorption are relatively insignificant. Lindane may exhibit slow soil release characteristics in various soils (i.e., soil desorption of the chemical is the controlling influence for dermal absorption), which limits the amount of dermal absorption predicted by the models.

Alternatively, Shatkin et al. (2002) developed a two-stage fugacity-based model specifically for BaP that incorporated both a fast soil desorption phase and a slow desorption phase of BaP from soil. Based on the several parameters investigated that would affect dermal bioavailability, the authors predicted that the fast desorption kinetics of a soil had a greater impact on predicted dermal uptake than any other parameter, including organic carbon content of a soil.

These examples show that the effect of soil on the dermal uptake of organic compounds can be difficult to predict without experimental data. However, dermal absorption by metal salts can be expected to be a more complex process than dermal absorption of organic compounds. Factors affecting absorption of soil-bound metals include pH, metal oxidation state, counter ion, size and solubility (Hostynek, 2003). For example, lead becomes more soluble and available for uptake in soil at low pH. However, a low soil pH tends to convert chromium (VI) to the larger less permeable chromium (III) ion. This reduction in chromium valence can also occur in transit through the skin and considerably slow the absorption of chromium through skin.

F.2.4 Effect of soil organic content on dermal absorption

For the soil pollutants discussed in this section, one of the most common soil variables explored for effect on dermal absorption of a chemical is the organic carbon or organic matter content. The chemical adsorbed to the organic carbon phase will generally be less available for transfer to skin than neat chemical present in a separate liquid phase in the soil, largely due to strong adsorption of the chemical to the organic carbon fraction (Bunge and Parks, 1996). Dermal bioavailability of a chemical in soil also tends to decrease with increasing organic carbon content of the soil (Sheppard and Evenden, 1994; Bunge and Parks, 1997). Consequently, a number of studies compared the effect of varying the

soil organic content on the dermal absorption of a chemical. The health protective approach for estimating an ABS would be to base the value on the higher dermal absorption from these studies, often from the soil with lower organic carbon content.

The length of time required for a chemical to partition to the soil organic material may be quite short (a few days) or longer (more than a month), depending on the nature of the deposited chemical, the soil and the weather (Bunge and Parks, 1996). However, early dermal absorption studies of chemicals in soil were usually conducted with freshly spiked soil just prior to exposure. Regardless of the partitioning time to the soil organic carbon, addition of a chemical to soil can often result in a reduction of dermal bioavailability relative to the pure chemical. For a group of selected organic compounds (e.g., DDT, BaP, PCBs, etc.) and arsenic, addition to soil just before loading onto skin reduced the overall dermal uptake by an average of about 60% compared to dermal uptake of the pure chemical (Wester and Maibach, 1999). However, a reduction in absorption from soil relative to a neat solution cannot be predicted for all chemicals. Dermal absorption for some chemicals such as arsenic in soil was found to be essentially unchanged compared to absorption from the neat solution.

F.2.5 Soil aging effects

The ABS point estimates presented here are primarily based on soils that were freshly spiked with contaminants and placed on skin for roughly 24 hrs. As such, the ABS point estimates largely represent the initial fast phase of decreased bioavailability when a chemical is freshly added to soil prior to skin exposure (Alexander, 1995; Bunge and Parks, 1997). This phase is generally a reversible process, such that a chemical sorbed to soil may become desorbed and be available for uptake during the skin exposure.

However, over time many chemicals added to soil undergo a slower second phase of decreased bioavailability. The soil-deposited chemicals tend to move from the external surface of soil particles to internal and more remote sites within the soil matrix so that chemicals become increasingly more desorption-resistant, a process known as aging (Alexander, 1995). A number of recent dermal absorption studies discussed below have observed reductions in dermal absorption occurring for up to 3-6 months following addition of the chemical to soil. Reductions of about 50% have been observed for dermal absorption of BaP aged in soil compared to soils freshly spiked prior to skin application (Roy and Singh, 2001). Abdel-Rahman et al. (1999) observed up to a 7.5-fold reduction in dermal absorption for arsenic aged in soil.

The continuous input of chemicals deposited on soils in the vicinity of “Hot Spots” stationary sources will likely result in the less recently deposited chemicals undergoing soil aging. For toxic inorganic metals in soil, the dermal dose equation (Eq. 6.1) does not account for decreased bioaccessibility over time due

to soil ageing. Leaching and weathering effects are assumed to be very long (i.e., 10^8 days), unless site-specific information shows otherwise. Only a few studies have investigated the decrease in dermal absorption for specific inorganic metals and metalloids aged in soils, including arsenic, nickel and mercury. The soil aging results from these studies are considered in the development of the ABS, although the volume of literature available is sparse. Therefore, dermal fractional absorption still relies primarily on data for freshly applied metals to soil to avoid underestimation of the ABS.

For organic chemicals, the soil half-life variable in Eq. 6.2 will account to some degree for the effects of soil aging, depending on the rigor of the extraction process used (Abdel-Rahman et al., 2002). Use of a strong acid extraction method may solubilize some of the desorption-resistant chemical from soil and overestimate the dermal bioaccessibility of a soil-aged organic chemical. That is why milder extraction methods have been recommended, such as soil extraction in synthetic sweat, to obtain a more applicable estimate of soil half-life.

F.2.6 Dermal soil loading and adherence characteristics

The ABS from soil depends on the amount of soil in contact with the skin. Maximal fractional absorption of a soil-bound chemical occurs when a monolayer of soil covers the skin (monolayer threshold). A monolayer can be defined, in this case, as layer of soil on the skin equal in thickness to the average soil particle diameter. Theoretical calculations and experimental data show that increased soil loading ($\text{mg soil}/\text{cm}^2$ skin) beyond monolayer coverage usually leads to decreased fractional absorption as a result of some of the soil not being in direct contact with skin (McKone, 1990; Duff and Kissel, 1996; Bunge and Parks, 1997). Soil loading at which the monolayer exists depends on the soil particle size (Duff and Kissel, 1996). For example, sand with an average particle diameter of 0.044 cm reaches monolayer coverage at $61 \text{ mg}/\text{cm}^2$, whereas monolayer coverage with clay at a particle diameter of 0.0092 cm is $13 \text{ mg}/\text{cm}^2$ (USEPA, 2004).

Early soil loading experiments were carried out under conditions of high loading, e.g. $20\text{--}40 \text{ mg}/\text{cm}^2$ (Shu et al., 1988; Wester et al., 1990a; Wester et al., 1992), without estimating monolayer coverage or providing average soil particle diameter to estimate monolayer coverage. High soil loadings that are greater than monolayer coverage may underestimate the fraction of chemical absorbed from soil. Coarse grain size (180 to 300 μm) used under the high loading conditions of $20\text{--}40 \text{ mg}/\text{cm}^2$ was at, or only, slightly more than monolayer coverage (Duff and Kissel, 1996). However, using such soil loadings with soils sieved to $<150 \mu\text{m}$ would result in greater than monolayer coverage.

Typical soil loadings under most human exposure scenarios generally ranged from 0.01 to $0.2 \text{ mg}/\text{cm}^2$ when averaged over the entire exposed skin surface (USEPA, 2004). Soil loadings on the hands, the skin region with the highest soil

loading, averaged about 1 to 5 mg/cm² during typical human activities in wet soil with a moisture content of 9 to 18%, and usually less than 0.1 mg/cm² with activities in dry soil with a moisture content of 3-4% (Kissel et al., 1998a).

During dermal absorption studies, the soil used to measure dermal uptake is applied to the skin as a "dry" formulation, i.e. the solvent used in the preparation of the chemical laden soil, is allowed to evaporate prior to dermal application. The uptake of a soil-bound chemical from wet soil is expected to exceed the uptake from dry soil because of the increased humidity and temperature at the skin surface (Wester and Maibach, 1983). Such conditions exist for human exposure scenarios that involve high humidity, high temperature, and skin covering (e.g. gloves and clothing). Some studies are carried out under condition of occluded skin, and these studies could be used to estimate chemical absorption from soil when moisture is present.

In addition, the particle size distribution of soil adhering to skin also needs to be considered in dermal absorption studies. Most recent dermal absorption studies have sieved soil down to <150 µm prior to spiking with chemical and applying to skin. Studies have shown that soil particles in this size range tend to adhere to skin to the greatest extent (Driver et al., 1989; Sheppard and Evenden, 1994; Kissel et al., 1996a). In hand press studies by Kissel et al. (1996a), small particles ≤150 µm were found to adhere preferentially over larger particles ≥250 µm in dry soils of <2% moisture. Adherence in wet soils (12-18%) was roughly proportional to the soil particle size distribution of the original soil, although no consistent adherence was seen with soil moisture and particle size with five soils studied. Monolayer coverage with soil sieved to <150 µm will vary depending on the particle characteristics, but was shown in one instance to be about 2 mg/cm² with an estimated mean grain size of 12 µm (Duff and Kissel, 1996).

Choate et al. (2006) found that the dermally adhered fractions of two soil samples with wide distributions of particle sizes generally consisted of particles of diameters <63 µm or <125 µm, depending on the soil sampled. Adherence was similar whether the soils were applied dry (1.58-1.85% moisture) or moderately moist (3.35-3.81% moisture). With increasing moisture content of roughly 10% or greater, adherence increases significantly and a greater proportion of larger soil particles >150 µm are represented in the adhered soil (Holmes et al., 1996; Kissel et al., 1996a; Choate et al., 2006). Smaller adhering soil particles can be considerably different in composition, especially in organic carbon content, from larger particles that tend to stick to skin in less abundance. However, organic carbon content does not appear to enhance the adherence of any particle sizes (Holmes et al., 1996; Choate et al., 2006).

F.2.7 In Vivo Vs. In Vitro Experiments

It is generally recognized that the most reliable method for assessing skin absorption of a chemical is to measure penetration in vivo using the appropriate

animal model or human volunteers (Kao, 1990). Thus, in vivo data are preferred over in vitro data for determination of a chemical ABS in this exposure assessment. In vivo data may be lacking for some chemicals of interest in this document due to economical considerations for conducting tests in humans and other mammalian species, or due to ethical concerns for testing in humans.

In vitro studies have the benefit of measuring dermal absorption under more easily controlled environments. Human skin can be tested without the inherent risks of a clinical study, and absorption through skin and retention in skin can be directly measured. Consequently, in vitro dermal absorption studies are frequently performed and provide the basis for an ABS for some chemicals presented in this section, following careful consideration for relevance to in vivo human exposure.

Although good agreement has been found when comparing in vivo and in vitro absorption results for some chemicals, trends towards lower absorption with in vitro exposure have been observed. For example, lipophilic compounds frequently have limited solubility in the buffered aqueous receptor fluids often used for in vitro cell systems, impeding the flow into the receptor fluid and resulting in an underestimation of skin penetration (Wester and Maibach, 1999). In vivo, lipophilic compounds penetrate the stratum corneum and diffuse through skin and, because of the solubilizing and emulsifying abilities of biological fluid, may readily be taken away by the blood in the dermal vasculature.

A reduction in skin viability of excised skin samples may occur due to storage conditions prior to use and may affect dermal absorption measurements. For example, the metabolic properties of human skin are reduced if the skin samples were previously frozen. Some polycyclic aromatic compounds (PAHs) undergo extensive percutaneous metabolism when absorbed, and reducing the metabolic capabilities of skin samples will reduce dermal penetration of absorbed PAHs (Kao et al., 1985; Ng et al., 1992; Moody et al., 2009a).

For metal salts, it has been postulated that low diffusion values through the stratum corneum in vitro is a result of skin shunts (e.g., hair follicles and sweat ducts) swelling shut upon hydration of skin samples (Tregear, 1966; Hostynek, 2003). Skin shunts that bypass the stratum corneum are thought by some to be a significant absorption route for charged metals. For example, dermal absorption of nickel salts shows there is a surge in diffusion at the earliest stage, which then rapidly decreases towards steady state (Tanojo et al., 2001). The decrease in diffusion rate has been proposed to be a result of the skin tissue becoming hydrated, shutting down the skin shunts.

A further potential limitation under in vitro conditions is that diffusing compounds must traverse the epidermis and the entire dermis in order to reach the receptor fluid. In vivo, the majority of the absorption into the cutaneous microcirculation is thought to occur in the upper dermis and the penetrant compounds may not have

to diffuse across the entire thickness of the dermis. However, the bulk of the connective tissue in the dermis is often eliminated from the skin preparation by cutting the skin parallel to the skin surface with a dermatome (Poet and McDougal, 2002).

In vivo studies are not without limitations. Dermally applied chemicals are often radiolabeled to facilitate quantification of the usually low absolute amounts of chemical dermally absorbed. In small mammals, a total accounting of all dermally absorbed radioactivity can be estimated from excreta, carcass, and site of skin absorption. However, in larger mammals measurements of radiotracer are quantified in excreta and measurements from intravenous, intramuscular, or oral dosing are applied as a correction for tissue absorbed chemical. The validity of this method depends on the underlying assumption that metabolism and disposition of the applied compound is route independent, and that the pharmacokinetic behavior of the intravenous and topical doses is similar (Kao, 1990).

F.2.8 *Inter- and Intra-Species Specificity*

The variability in dermal absorption of chemicals among mammalian species has been investigated in vivo and in vitro. Bartek et al. (1972) suggest that the extent of in vivo uptake among animals follows the rank: rabbit > rat > pig \approx monkey \approx humans, based on dermal absorption of benzoic acid, hydrocortisone, testosterone, caffeine, N-acetylcysteine, and butter yellow. However, the species ranking did not strictly hold for all chemicals, indicating not only species-specific differences but also chemical-specific differences.

Comparison of data from other studies does support that in general, the absorption in the rabbit, rat and other rodents can considerably overestimate absorption in humans, while absorption in monkeys and miniature pigs most closely predict human absorption (Wester and Maibach, 1975; Reifenrath et al., 1984; Wester and Maibach, 1985; Bronaugh et al., 1990; Wester et al., 1998a). Alternatively, Kao et al. (1985) found that in vitro permeation of testosterone and BaP through human skin was greater than that for guinea pig, rat, or rabbit, indicating that species-specificity differences likely depends on other factors such as experimental conditions and tissue viability. Variability in dermal absorption depending on the skin area exposed has been investigated (Wester and Maibach, 1983). In humans, absorption across the skin varies by area of the body and may be higher than the commonly used forearm (e.g. scalp, axilla, forehead, jaw angle and scrotum).

F.2.9 *Metabolism of Absorbed Chemicals in the Skin*

The description of percutaneous absorption is generally based on diffusion models that take into account the physico-chemical characteristics of chemicals and soils. While such descriptions may help to explain the uptake of chemicals

across the stratum corneum, the role played by metabolism in the viable epidermal and dermal layers should be included to understand the complete permeation of chemicals through the skin (Wester and Maibach, 1983; Kao and Carver, 1990; Bronaugh et al., 1994).

Viability of the skin refers to the status of active energy turnover, i.e. the utilization of glucose and formation of CO₂ or lactate in skin. Enzymes and metabolic processes in skin may affect the dermal penetration of drugs and other xenobiotics, particularly if absorbed chemicals can be metabolized in the skin. Using production of lactose as the measure of viability, human skin placed in a buffered solution and kept refrigerated remained viable for about 8 days following donor death (Wester et al., 1998b). Skin frozen for storage or heat-treated to separate the epidermis and dermis renders the skin non-viable and may change the dermal penetration dynamics of absorbed chemicals. Some early studies investigating the dermal penetration of chemicals used previously frozen skin samples and may not provide a good basis for ABS determination.

Dermal metabolism of BaP was observed to be considerably reduced in several mammalian species with use of non-viable skin, resulting in reduced penetration of BaP through skin (Kao et al., 1985). In viable human skin, nearly half the BaP that permeated the skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid. In fact, dermal absorption of polycyclic aromatic hydrocarbons (PAH) that include BaP resulted in PAH-DNA adducts in human skin samples, demonstrating that skin is a target organ due to metabolic activation of PAHs in skin (Phillips et al., 1990).

On the other hand, dermal absorption of some chemicals does not appear to be affected by the viability status of the skin samples. Dermal penetration of TCDD through viable and non-viable pig skin was found to be similar (Weber, 1993).

F.2.10 Human Adult and Infant Variability in Skin Permeability

Animal studies are designed to ensure uniformity within the experimental population by using inbred strains and often only one sex. The variability between animals is much less than the genetically diverse human population. Human studies also rarely use children or infants, the elderly, pregnant women and the infirm, partially because of ethical considerations. Dermal uptake may vary due to genetic diversity in the human population and differences in age. This variability will not necessarily be accounted for by experimental data.

A review of the data on human skin permeability to chemicals suggest at least a mean intra-individual coefficient of variation of approximately 40% and a mean inter-individual variation of about 70% (Loth et al., 2000; Hostynek, 2003). A leading cause in the variation is the lipid composition of the stratum corneum, which influences solubility and permeability of drugs. This factor is partly

responsible for the high variability in accumulation and permeation measurements (Loth et al., 2000).

There has been increasing awareness in recent years that infants and children are more susceptible than adults to the harmful effects of some pollutants. This can be due to differences in exposure, physiology, absorption, distribution, metabolism, and excretion. Further, organ development and faster cell division influences targets of toxicity. Finally, a large skin surface area to body weight ratio would increase the dose of an absorbed chemical on a mg/kg body weight basis.

Only a few studies have examined age-related differences in the dermal absorption capacity of chemicals in infants and children compared to adults. Preterm infants lack a fully developed dermal barrier function and are particularly prone to accidental poisoning of toxic agents applied to the skin surface (Barrett and Rutter, 1994). In an in vitro system, McCormack et al. (1982) observed increased penetration of some alcohols and fatty acids through skin of premature infants compared to full term infant skin and adult skin. Dermal absorption of sodium salicylate was found to be a hundred- to a thousand-fold greater in infants of 30 weeks gestation or less compared to full term infants (Barker et al., 1987).

In full-term infants, epidermal structure and function matures by 2-3 weeks of age (Holbrook, 1998; Makri et al., 2004). In general, the in vitro system of McCormack et al. (1982) showed full-term baby skin to be a good barrier for some compounds. No difference in penetration of alcohols through full term infant and adult skin was seen. However, penetration of some fatty acids through full term infant skin was greater than that through adult skin. Higher lipid content in the stratum corneum of infants was thought to be the reason for increased absorption of fatty acids. In addition, a layer of subcutaneous fat develops at approximately 2-3 months of age in infants and continues to exist through the early toddler period (Thompson, 1946; Banks et al., 1990; Cohen Hubal et al., 2000). This layer of fat may act as a sink for lipophilic chemicals absorbed through the skin.

Age-related changes in dermal absorption have also been investigated in experimental animal models. Using TCDD or 2,3,4,7,8-pentachlorodibenzo-p-dioxin (4-PeCDD) in solvent, Banks et al. (1990) observed greater absorption of TCDD or 4-PeCDD in 10-week old rats than 36 - 120-week old rats. 2,4,5,2',4',5'-Hexachlorobiphenyl showed significantly higher fractional penetration in young rats (33 days old) compared to adult rats (82 days old) in vivo, but only at one of three dose levels tested (Shah et al., 1987). Overall, the authors concluded that no clear age-related pattern of dermal absorption was found among a total of 14 pesticides including 2,4,5,2',4',5'-hexachlorobiphenyl.

F.2.11 Use of Default ABS Values

The California South Coast Air Quality Management District's Multi-Pathway Health Risk Assessment Input Parameters Guidance Document (SCAQMD, 1988) recommended using default values of 10% for organic chemicals and 1% for inorganic chemicals when quantitative data are not available to estimate chemical-specific dermal absorption fractions from soil.

Use of these default factors was proposed based on a review of the dermal absorption literature and recommendations by McLaughlin (1984). In his US EPA report, McLaughlin suggests it may be possible to group penetrants into a numerical system using an "order of magnitude" approach (i.e., 100% - 10% - 1% - 0.1% fractional absorption groupings), depending on physical parameters such as partition coefficients and diffusion constants. For example, many of the organic compounds were found to fall into the 10% absorption range. Exceptions included some pesticides, such as the very lipophilic pesticide carbaryl that exhibited a fractional absorption closer to 100%, and the polar pesticide diquat that exhibited a fractional absorption closer to 1%.

More recently, US EPA (2004) also recommended a default dermal absorption fraction for semivolatile organic compounds (SVOCs) of 10% as a screening method for the majority of SVOCs without dermal absorption values. This fraction was suggested because the experimental values for SVOCs determined by US EPA are assumed to be representative of all SVOCs as a class. US EPA (2004) notes that chemicals within classes can vary widely in structure and chemical properties, potentially resulting in a wide range of fractional absorption values. However, OEHHA agrees that a 10% fractional absorption default value is acceptable at this time, based on the range of values (3 to 14%) estimated in Table F.5 for SVOCs.

For inorganic classes of compounds, US EPA (2004) recommended no default dermal absorption values be used. The premise was that speciation of inorganic compounds is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value. With the exception of the metalloid arsenic, OEHHA notes that the range of ABS point estimate values for inorganic metals (see Table F.5) have a relatively narrow range, between 0.2 and 4%. Therefore, it is reasonable to assume that a default ABS of 1% can be used as a screening value, primarily if there are some data to indicate that the metal salt exhibits characteristics of low fractional dermal uptake similar to other metal salts. Currently, OEHHA default ABS values apply only to inorganic compounds of fluoride, beryllium and selenium.

F. 3 Point Estimates for Dermal Absorption (ABS) of Inorganic Compounds

F. 3.1 Arsenic and Arsenic Compounds

Recommended point estimate for dermal uptake: 6%.

F.3.1.1 Studies Considered

A. Key Studies

Wester et al. (1993a) examined the in vivo percutaneous absorption of radiolabeled soluble arsenic (as $\text{H}_3^{73}\text{AsO}_4$) freshly mixed with soil and applied to skin of female Rhesus monkeys ($n = 4$ animals per dose group). Dose levels of 0.0004 and 0.6 $\mu\text{g}/\text{cm}^2$ were used. The soil load on the skin was 40 mg soil/ cm^2 skin area. The soil had been sieved to 180-300 μm prior to application, thus, a soil load of 40 mg/ cm^2 was likely at or near monolayer coverage. Topical doses were applied to an area of the abdomen for 24 hours. Urine was collected during the dosing period, and through the following 6 days. For comparison, radiolabeled arsenic (as ^{73}As) in water was administered intravenously to four monkeys. Percutaneous absorption was determined by the ratio of urinary arsenic excretion following topical application to that following intravenous administration.

Urinary excretion of the ^{73}As label was complete by day 7, with about half the label excreted in the first 24-48 hrs following topical administration. Results of this study showed that the percutaneous absorption of arsenic from soil was $4.5 \pm 3.2\%$ from the low dose and $3.2 \pm 1.9\%$ from the high dose (nonsignificant difference). An estimate of arsenic retained in the skin was not performed, although 27-28% of the arsenic could not be accounted for following decontamination of the skin.

Lowney et al., (2005) conducted follow-up absorption studies with arsenic aged in soil that paralleled the methodology used in the in vivo Rhesus monkey study. The soil samples collected were adjacent to a pesticide production facility that had historically produced calcium and lead arsenate compounds. The arsenic was resident in the soil for a minimum of 30 years and was primarily in the sparingly soluble iron oxide and iron silicate mineral phases. Small amounts of more soluble calcium arsenate and arsenic trioxide were also detected in the soil. The particle size fraction was sieved to $<150 \mu\text{m}$ and a skin loading of 4 mg/ cm^2 on 100 cm^2 of skin was applied. Total dose was 560 μg arsenic and the duration of dermal exposure was 8 hrs on the abdomens of three monkeys. Following fractional correction of arsenic from i.v. dose, urinary excretion of arsenic ranged from 0.01 to 0.24% of the dermally applied dose, but was not statistically greater than background. Negligible absorption was considered due to the presence of

soil arsenic primarily in sparingly soluble mineral phases. Direct or indirect estimates of arsenic retained in the skin were not performed.

A sweat extraction technique by Nico et al. (2006) was employed to estimate the soluble arsenic that can be made bioavailable for dermal absorption from the aged arsenic soil used in the in vivo monkey study by Lowney et al. (2005). Sweat extraction of this soil resulted in only 1.8% soluble arsenic. However, a second aged soil sample from a different arsenic-contaminated site resulted in 11% arsenic extracted by sweat. Nico et al. (2006) also used the sweat extraction technique to estimate soluble arsenic from soil samples freshly spiked with arsenic. One sample was sieved to <150 μm while another was sieved to 180-300 μm , similar to that used by Wester et al. (1993a) in the in vivo dermal monkey study. Sweat extraction resulted in 45 and 72% soluble arsenic from the <150 and 180-300 μm soil samples, respectively.

B. Supporting Studies

In addition to the monkey in vivo study, Wester et al., (1993a) conducted an in vitro study using human cadaver skin from three separate donor sources with three replicates from each source. The skin was dermatomed to 500 μm , stored refrigerated in Eagle's medium and used within 5 days to preserve skin viability, although elapsed time from death to harvest of skin was not specified. A dose of 0.0004 μg arsenic per cm^2 skin surface exposed was applied. The soil load on the skin samples was 40 mg soil per cm^2 skin area, and phosphate-buffered saline served as receptor fluid. The in vitro exposure period was 24 hours. As performed in the monkey in vivo study, the soil had been sieved to 180-300 μm prior to application, so monolayer coverage was probably not surpassed. Percutaneous absorption through human cadaver skin was 0.76% (0.43% in receptor fluid; 0.33% in skin) after soap and water wash. While the authors did not speculate as to the reduced in vitro dermal absorption compared to monkey in vivo absorption, Kao (1990) noted that both elapsed time from death to harvest of tissues and treatments and storage of the cadaver could have resulted in a large variability in skin permeability.

Dermal absorption of radiolabeled soluble arsenic (as $\text{H}_3^{73}\text{AsO}_4$) freshly applied or aged in two different soils was determined in vitro through dermatomed pig skin cut 200 μm thick (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter, with no apparent sieving before application. Arsenic was applied to skin for 16 hrs either alone in ethanol vehicle, immediately after the addition of 30 mg of the soils to skin, or after aging for 3 months in each soil. Soil loading was calculated to be about 47 mg/cm^2 . Applying soil to skin and then applying the arsenic does not allow time for arsenic-soil equilibrium. This method of application allows for direct contact of skin with arsenic or vehicle and not from soil, leading to an overestimation of the fractional absorption (Spalt et al., 2009).

In addition, monolayer coverage was probably exceeded with a soil loading of 47 mg/cm².

With arsenic freshly added to soil, 0.2% of the arsenic penetrated the skin to receptor fluid from both soil types (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999). Total dermal absorption including arsenic retained in skin was 10.0 and 6.0% from the sandy and clay soils, respectively. In comparison, pure arsenic found in receptor fluid and retained in skin was 0.4 and 44.2%, respectively. In aged sandy and clay soil, 0.2 and 0.1% arsenic was found in the receptor fluid, respectively. Total dermal absorption in the aged soils was 1.5 and 0.8% from sandy and clay soils, respectively.

Radiolabeled sodium arsenate was applied in vitro to the skin of mice for 24 hrs as a solid compound, in an aqueous solution, or as an aqueous solution in sandy soil (Rahman et al., 1994). Soil was sieved to $\leq 180 \mu\text{m}$ and contained 58% sand, 34% silt, 8% clay and 1.4% organic matter. Arsenate was freshly applied to soil prior to skin application, with an average soil loading on the skin of 23 mg/cm². Absorption increased linearly with the applied dose from all exposure vehicles, with a constant fraction of the dose being absorbed. Total arsenate absorption was as high as 62% of applied dose from 100 μl water vehicle and about 33% of applied dose as the solid. However, absorption of arsenate from soil was less than 0.3% of applied dose, with about one-third penetrating to the receptor fluid.

A dermal exposure study was conducted to assess the potential for arsenic exposure in children in contact with playground equipment and decks treated with the wood preservative chromated copper-arsenate (CCA) (Wester et al., 2004). Methodology was similar to that used by Wester et al. (1993a) in three monkeys to assess dermal arsenic absorption from CCA-treated wood residues. Following 8-hr dermal application, an increase in urinary excretion of arsenic above background was not detectable, indicating virtually no absorption of arsenic from CCA-treated wood residue. The researchers determined that the absorbed dose would need to be in the range of 0.10 to 0.16% of the applied dose to be detectable above background.

The negligible dermal absorption of arsenic from the CCA residues is a result of arsenic chemically bound with other metals (particularly chromium) and ultimately to the wood structure (Nico et al., 2004). The leaching characteristics of soluble arsenic in CCA residues were also investigated by extraction in human sweat (Nico et al., 2006). The sweat extraction procedure indicated that up to 12% of total arsenic is available for dermal absorption from CCA-treated wood residue. However, only 1.4% soluble arsenic was extracted with sweat from CCA-residue aged in soil near a CCA-treated utility pole. Gastric leaching conditions resulted in up to 2-3 times greater solubilization of arsenic from CCA-treated wood compared to sweat leaching, indicating soil ingestion of CCA-released arsenic can be a health concern.

F. 3.1.2 Discussion and Recommendation for Arsenic and Arsenic Compounds ABS

Dermal exposure of skin to arsenic resulting in passage of arsenic through skin to the bloodstream is the primary concern under the “Hot Spots” program. However, arsenic that becomes bound in skin may also have toxicological consequences. Regardless of route of exposure to arsenic the skin is a critical target organ for arsenic toxicity due to local absorption and binding of sulfhydryl-group-containing proteins (Hostynek et al., 1993). The affinity for sulfhydryl groups leads to arsenic’s accumulation and tenacious retention in keratin-rich tissues such as hair, nails, and skin. Measurement of in vitro percutaneous absorption of As(III) and As(V) by human epidermal skin cultures for 6 hrs shows strong affinity of arsenic for the keratinocytes, with an estimated 30% of As(V) passing through skin being retained compared to over 90% of the As(III) being retained (Bernstam et al., 2002).

Accumulation of arsenic in the skin is characterized by hyperpigmentation, keratoses of the palms of the hands and soles of the feet, and diffuse macular pigmentation or diffuse darkening of the skin on the limbs and trunk, attributed to the reduction and deposition of the element in the metallic state (Hostynek, 2003). Chronic arsenic accumulation in skin increases the susceptibility of the skin to ultraviolet light and is associated with an increased incidence of tumors of exposed skin, although skin cancer is primarily a result of oral arsenical poisoning and characterized by multifocal lesions over the entire body (Hostynek et al., 1993; OEHHA, 1999).

The key in vivo monkey study by Wester et al. (1993a) provides an average fractional absorption of 3.9% based on two dose levels of arsenic that had been freshly added to soil before application to skin. Some limitations are noted for this study. First, the in vivo study did not estimate arsenic retained in skin. However, the researchers followed excretion of arsenic after exposure and noted that excretion of the labeled arsenic was essentially over by day 7. The remaining arsenic bound to skin proteins will probably remain there and not present a risk of reaching the bloodstream.

Secondly, a sieved soil fraction of 180-300 μm was used, which does not reflect the generally smaller soil particle fraction that sticks to skin following dermal contact. Soil sieved to <150 μm is considered more relevant for dermal studies (Spalt et al., 2009). The sieved soil used by Wester et al. may underestimate fractional absorption. This assumption is supported by the sweat extraction study by Nico et al. (2006), which found a 63% increase in arsenic bioavailability (45% to 72%) from soil sieved to <150 μm as opposed soil sieved to 180-300 μm .

Finally, there is also some question whether the contaminated soil had continuous contact with the skin of the monkeys (Spalt et al., 2009). From the methodology description, the eye patches used to hold the soil in place on the abdomen of the monkeys were a larger volume than the applied soil. Thus, sloughing of soil off the skin probably occurred when the monkeys sat upright.

Together, these limitations indicate that basing an ABS on the monkey study may underestimate the dermal fractional absorption of arsenic. However, the sweat extraction study by Nico et al. (2006) supports the application of an adjustment to account for use of a soil fraction that likely underestimates fractional absorption. A 63% increase in arsenic bioavailability was observed from soil sieved to <150 μm , compared to soil sieved to 180-300 μm , as used by Wester et al. (1993a). A soil sieved to <150 μm better characterizes the soil particle size that adheres to skin. Thus, a 63% increase was applied to the monkey fraction absorption value of 3.9% resulting in an arsenic ABS of 6% when rounded to the nearest whole number.

The in vitro studies reviewed here gave a range of 0.3 to 10% for total absorption following application of freshly spiked soil to skin samples (Rahman et al., 1994; Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999; Wester et al., 1993a). However, arsenic aged in two soils gave a total dermal absorption of 0.8-1.5% in pig skin in vitro (Abdel-Rahman et al., 1996). As discussed above, it is difficult to reconcile the difference in dermal absorption in pig skin between arsenic freshly spiked in soil and arsenic aged soil due to differences in methodology. Future in vitro studies using human skin and arsenic freshly applied and aged in soils would help assess the impact of arsenic aged in soil.

F. 3.2 Beryllium and Beryllium Compounds

Recommended use of default inorganic compound ABS estimate of 1.0%.

F. 3.2.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption or skin penetration of beryllium (Be) compounds. Be metal powder can oxidize when suspended in synthetic sweat, whereupon the metallic ions may be absorbed in human skin (Larese et al., 2007). However, Be salts are corrosive to skin, and have a high reactivity with protein substrates that result in strong retention in skin (Hostynek et al., 1993). The reaction of beryllium salts with the proteins in skin acts as a strong sensitizer that cause allergic contact dermatitis. Beryllium compounds typically decompose to form the poorly soluble, amorphous oxide (BeO) or hydroxide (Be(OH)₂), resulting in tissue granulomas (i.e., compactly grouped cells that replace normally functioning tissue) and ulcers. Once lodged in tissue, these amorphous beryllium precipitates are excreted at a very slow rate.

Belman (1969) investigated the interaction of beryllium fluoride and beryllium sulfate with guinea pig epidermal tissue in order to explore a mechanism for the delayed allergic skin reaction observed in humans following beryllium exposure. Using both in vitro and in vivo experiments, he reported that beryllium is taken up into the skin and localized primarily to proteins of the epidermis, with little or no apparent binding to stratum corneum or dermis. Exposure caused a localized immune response and rapid destruction of skin cells. Data are not provided, however, regarding the amount of beryllium taken up by the skin cells, or the fate of beryllium following the immunological response (i.e., whether beryllium is then absorbed into the circulation, or sloughed off with cells.)

Petzow and Zorn (1974) reported on the absorption of beryllium through the tail skin of rats exposed to an aqueous beryllium chloride solution spiked with ^7Be . The authors stated that within the first hour of exposure there is an increase in the rate of beryllium uptake. After approximately 90 minutes, the dermal flux of beryllium from the aqueous solution is constant. In addition, Petzow and Zorn reported that the amount of beryllium that diffuses through the skin seems to be dependent upon the concentration of beryllium in contact with the skin.

Worker exposure and likely facility emissions of beryllium compounds are mostly in the form of particulates, primarily BeO (Tinkle et al., 2003; Day et al., 2006). For these poorly soluble beryllium particles, dermal exposure is considered to be of toxicological significance. Chronic beryllium disease (CBD) is an occupational disease that begins as a cell-mediated immune response to inhaled beryllium. Although respiratory and engineering controls have significantly decreased occupational inhalation exposures, reduction in occurrence of beryllium sensitization and CBD has not significantly decreased. The lack of worker skin protection has been postulated as a contributor to the persistence of sensitization and CBD in the workplace.

The concentration of antigen required for elicitation of a cell-mediated immune response is significantly smaller than the concentration required for sensitization, therefore, the failure of respiratory exposure limits to lower the rate of disease is likely related to the continued unchecked skin exposure to beryllium particles (Tinkle et al., 2003; Day et al., 2006; Deubner and Kent, 2007). Thus, in workers with significant beryllium skin exposure, the pulmonary exposure required to elicit a subsequent immune response and granuloma formation would be significantly smaller.

To determine if BeO can penetrate the stratum corneum and reach the immunologically active epidermis, Tinkle et al. (2003) conducted a pilot study in which BeO particles were suspended in petrolatum (1 mg/g), painted on the back of shaved mice, and the area covered with surgical tape. The average amount of beryllium applied to each mouse was 70 μg . Excess BeO was removed from the surface of the flank skin by gentle washing and tape stripping three times immediately following 24-hr exposure. On day 7 or 14 following the exposure,

the amount of beryllium in the flank skin of BeO-treated mice was, on average, 1.2 µg/g tissue, thus confirming that BeO is present in the skin.

Additionally, Tinkle et al. (2003) observed in vitro that polystyrene latex spheres <1 µm in diameter, when applied to skin and coupled with flexing motion, can penetrate intact human skin. The researchers proposed that beryllium particles can similarly penetrate the skin.

F. 3.2.2 Discussion and Recommendation for the Beryllium and Beryllium Compound ABS

Due to the lack of quantitative data regarding dermal absorption of beryllium, it is not possible to calculate a chemical-specific fractional absorption value for Be salts. The high reactivity of beryllium with skin suggests penetration to the bloodstream in intact skin is small relative to other inorganic metals discussed in this section. However, it is postulated that a primary concern for dermal exposure to beryllium is related to sensitization, which results in much lower inhaled concentrations of beryllium particles required for elicitation of a cell-mediated immune response leading to progression of CBD (Tinkle et al., 2003; Day et al., 2006). This action only requires penetration to the epidermis where the immune response occurs. Considering full dermal penetration of beryllium to the bloodstream may not be required to enhance or facilitate a toxicological response, and that particles have been shown to penetrate the skin with flexing, it is recommended that a default ABS of 1% for inorganic beryllium compounds in soil be used for screening purposes to assess dermal exposure.

F. 3.3 *Cadmium and Cadmium Compounds*

Recommended point estimate for dermal uptake: 0.2%.

F. 3.3.1 Studies Considered

A. Key Studies

Wester et al. (1992) examined the percutaneous absorption of cadmium chloride from soil using human cadaver skin in an in vitro system. Donor skin was used within 5 days of harvest and was kept refrigerated in buffered medium until then. The soil used prior to sieving contained 26% sand, 26% clay, 48% silt and 0.9% organic carbon. The soil was sieved to retain particles in the range of 180 to 300 µm. Radiolabeled cadmium (¹⁰⁹Cd) was mixed with soil at a concentration of 13 ppb and applied to the skin samples at a soil loading of 20 mg/cm² or 40 mg/cm². Two donor skin sources were used with replicates for each of the soil concentrations. Human plasma was used as the receptor fluid. At the end of a 16-hour exposure, soil was removed from the samples by soap and water rinse. Percutaneous absorption, calculated as receptor fluid accumulation plus residual skin concentration after soap and water wash, ranged from 0.08% to 0.2% of

applied dose (Table F.1). No significant differences were observed in absorption between skin samples or soil load concentrations.

Table F.1. In Vitro Human Dermal Fractional Absorption of Cadmium Chloride from Soil^a

Soil Loading	Skin Source	Percentage Applied Dose		
		Receptor Fluid	Skin	Total
40 mg/cm ²	1	0.02 ± 0.01	0.06 ± 0.02	0.08
	2	0.07 ± 0.03	0.13 ± 0.05	0.20
20 mg/cm ²	3	0.02 ± 0.02	0.08 ± 0.06	0.1
	4	0.02 ± 0.02	0.08 ± 0.06	0.1

^a Data from Wester et al. (1992); n = 3 replicates per skin source

In a another experiment, Wester et al. (1992) applied cadmium in water to human skin samples for 30 min, followed by removal of the cadmium solution from the skin surface and continued perfusion of the skin for an additional 48 hrs. No cadmium appeared in the receptor fluid after 30 min of exposure. However, 0.6 ± 0.8% of the dose had diffused into the receptor fluid after 48 hrs demonstrating the capacity of cadmium to be retained in the skin and be slowly systemically absorbed over time.

B. Supporting Studies

Kimura and Otaki (1972) used liver and kidney accumulation of cadmium in rabbits and hairless mice to estimate dermal absorption. A total dose of 30.5 mg Cd (in an aqueous CdCl₂ solution) was administered to rabbit skin (n=1) in 5 doses over 3 weeks. Two weeks after the final application, 0.40% of the applied dose was found in liver and kidney combined. In rabbits (n=2), a total dose of 61 mg Cd was administered in multiple cream-like and milk-like ointment applications, resulting in 0.45 and 0.61% of the applied dose, respectively, in liver and kidney combined. The type of ointment vehicle used did not appear to greatly affect the absorption or accumulation characteristics of Cd. Dermal absorption of cadmium in hairless mice, estimated from kidney and liver accumulation, ranged from 0.07-0.27% after a single application of ointment (0.61 mg Cd). Cadmium absorption after multiple ointment applications on hairless mice ranged from 0.59 - 0.87% of applied dose.

Aqueous 1.0, 0.1 and 0.01% cadmium solutions were painted onto the skin of mice and rats and air dried each day for ten days (Lansdown and Sampson, 1996). Perceptible skin damage occurred at the two highest doses, likely resulting in increased dermal absorption. At the lowest dose, significantly

increased skin content of cadmium was observed in both mice (138 ng Cd/g) and rats (248 ng Cd/g). Adequate data to estimate fractional absorption were not provided.

Although no studies estimated dermal absorption of cadmium aged in soils, Aringhieri et al. (1985) reported that 80% of cadmium added to a soil containing high organic matter (14.2%) and high clay content (60%) was adsorbed to soil particles within 10 min of addition to a soil. Tang et al. (2006) observed that bioaccessibility of cadmium (relating closely to absorption following ingestion of soil) in strongly acidic soils spiked with cadmium reached nearly steady state levels as high as 77% after the first week of aging. In soils highly contaminated with heavy metals by industrial sources, the MgCl_2 -exchangeable fraction of cadmium was about 37% and was considered the most mobile and biologically available heavy metal in the samples examined (Hickey and Kittrick, 1984).

F. 3.3.2 Discussion and Recommendation for a Cadmium and Cadmium Compounds ABS

No in vivo studies investigating fractional absorption of cadmium from soil were located. The human in vitro study by Wester et al. (1992) provided the only quantitative data for dermal absorption of cadmium from soil. The retention and concentrating of cadmium in skin with slow systemic absorption demonstrate the necessity for including the cadmium found in exposed skin for estimating an ABS point estimate.

The lack of quantitative in vivo studies and the use of 16 hr rather than 24 hr exposures support a point estimate based on the highest fractional absorption of 0.2%, rather than a the lower estimate of 0.1% (based on an averaging of different skin sources for each of the two soil loadings). In addition, coarse particle soil loadings of 20 and 40 mg/cm^2 may result in a reduced fractional absorption, although the data suggest monolayer coverage of skin was probably not exceeded (Spalt et al., 2009). The high bioavailability and apparent low capacity for aging of cadmium in some soils indicates that sequestration of cadmium in soil will be small relative to other inorganic metals in soil.

F. 3.4 Soluble Compounds of Hexavalent Chromium

Recommended point estimate for dermal uptake: 2%

F. 3.4.1 Studies Considered

A. Key Study

Czernielewski et al. (1965) exposed guinea pigs to hexavalent chromium (chromium (VI)) as sodium chromate solution labeled with Cr^{51} . A single dose (15 μg sodium chromate in 0.1 ml solution) was applied to a 4 cm^2 shaved area

of skin for 24 hours (n=9 animals). Absorption was estimated by measurement of the Cr⁵¹ content of the following: urine, feces, blood (1 ml), heart, liver, spleen, adrenals, kidneys, lungs, lymphatics, and skin. Dermal absorption of chromium (VI) was estimated to be 2.9% of the applied dose from the 24 hour exposure. Based on the average blood volume of adult guinea pigs (27 ml), 1.6% of applied dose was found in blood, 1.1% in excreta, and only 0.2% in organs and tissues including skin.

B. Supporting Studies

Chromium in the hexavalent [Cr(VI)] state does not measurably bind with proteins, whereas the trivalent chromic ion [Cr(III)] shows strong affinity for protein in epithelial and dermal tissues (Samitz et al., 1969; Gammelgaard et al., 1992). Thus, Cr(VI) can permeate through skin relatively easily compared to Cr(III). However, skin has the capacity, though limited, to reduce Cr(VI) to Cr(III) resulting in binding of chromium to skin protein and decreasing the rate of diffusion (Gammelgaard et al., 1992; Hostynek, 2003). Binding of chromium in the skin is characterized as irreversible, leading to protein denaturation with formation of permanent depots in the epidermis (Hostynek, 2003). Some of the bound chromium is likely subject to the counter-current effect of continuous sloughing of the outer skin layers, although no studies have attempted to quantify this removal pathway.

To investigate the level of penetration of Cr(VI) into human skin, Liden and Lundberg (1979) cut 10 µm tangential sections of skin biopsies after application of a 0.5% aqueous potassium chromate solution on a 79 mm² patch of skin on the back of volunteers. Dermal exposure durations to the chromate were 5, 24, or 72 hrs. Highest chromium levels were found in stratum corneum. Chromium was also found at the dermal-epidermal junction and the upper mid-dermis. Chromium levels differed considerably between different biopsies, but the content of chromium was the same order of magnitude at all exposure durations indicating that a steady state was reached within 5 hrs of exposure.

Alternatively, Mali et al. (1964) measured the disappearance of a radiolabeled chromate solution absorbed dermally in two human volunteers and determined penetration into stratum corneum by tape stripping. Application of a 0.02 ml 0.25% dichromate solution (containing 50 µg Cr(VI)) on a patch to the arm for 12 hrs resulted in the disappearance, and presumed absorption, of 22 µg Cr into the skin. Tape stripping of stratum corneum removed 0.35 µg of radiolabel in the skin.

Systemic uptake of chromium was studied in four human volunteers following a three hour submersion in a tub of water containing 22 mg/L Cr(VI) as potassium dichromate (Corbett et al., 1997). Urinary chromium excretion showed large intra-individual variability. Five-day total Cr urinary excretion above historical background ranged from 17.5 to 1.4 µg, with an average of 6.1 µg. Urine levels

of chromium were normal in three volunteers by day 2, although a fourth volunteer excreted elevated levels of chromium up to the end of the experiment on day 5. Elevated blood and serum levels of chromium were recorded within 1 hr after end of exposure. Chromium content of red blood cells was generally increased about 2-fold, and serum content was increased about 3- to 5-fold. Chromium levels in red blood cells and serum had returned to control levels 2 days after exposure. The systemic uptake rate through skin ranged from $4.1\text{E-}04$ to $7.5\text{E-}05 \mu\text{g}/\text{cm}^2\text{-hr}$ with an average of $1.5\text{E-}04 \mu\text{g}/\text{cm}^2\text{-hr}$.

Aqueous solutions of Cr(VI) as potassium dichromate, and Cr(III) as chromium trichloride and chromium nitrate were applied in vitro to full thickness human abdominal skin in diffusion cells at a chromium content of 0.034 M (Gammelgaard et al., 1992). Test solutions of $556 \mu\text{l}/\text{cm}^2$ were applied over a skin surface area of 1.8 or 0.7 cm^2 . After 190 hrs exposure of skin to the dichromate, 134 and 12 $\mu\text{g Cr}/\text{cm}^2$ were found in the epidermis and dermis, respectively. Only 0.037 $\mu\text{g Cr}/\text{cm}^2$ was found in the recipient phase. A total Cr(VI) permeation of 15% was calculated. Significantly less Cr(III) from either the trichloride or nitrate was found in skin. Cr(III) content in skin was no more than 9% of the chromium content applied as Cr(VI), with no chromium found in the recipient phase. The lower permeation of Cr(III) was considered a result of the skin acting as a barrier to absorption of the positive Cr(III) ions.

In other experiments by Gammelgaard et al. (1992), application of the dichromate at concentrations of 0.125, 0.25, and 0.5% to skin for 48 hrs showed increased Cr content in skin with increasing concentration, although no Cr was detected in the recipient phase. Total percent Cr permeation of 0.7, 0.7 and 1.1% was calculated for exposure to the 0.5, 0.25 and 0.125% dichromate solutions, respectively. Increasing dichromate concentration (0.5 to 2.5% Cr solution concentrations) with 168 hr exposure did not result in increased Cr content in skin. Long lag times for appearance of Cr in the recipient phase combined with lack of increased skin concentration with time indicates a high binding capacity for Cr that will interfere with diffusion through the skin, although skin binding sites can eventually be exhausted with time. Gammelgaard et al. (1992) also observed the ratio of Cr(VI) to Cr(III) at pH 10 in the recipient phase to increase over 160 hr of exposure. Appearance of chromium as Cr(VI) in the recipient phase increased from about 60% at 40 hrs, to greater than 90% at 120 hrs. This finding indicated reduced capacity for dermal Cr(VI) reduction, eventually resulting in increased Cr(VI) passing through the skin.

Baranowska-Dutkiewicz (1981) found chromium (VI) from aqueous solutions to be readily absorbed by human skin. Seven volunteers were exposed to sodium chromate solutions (0.01, 0.1, and 0.2 M) on an area of the forearm for 15, 30 or 60 minutes, in a series of experiments. The exposure area was covered with a watch glass throughout the exposure period. Absorption was calculated from the difference between the applied and recovered dose of chromium (VI). The authors reported that percutaneous absorption of chromium is dependent on both

concentration and time. Specifically, they found that (1) absorption was highest from the 0.01 molar solution (7.7-23% of applied dose) and lowest from the 0.2 molar solution (3.4-10.6% of applied dose), (2) the rate of absorption decreased as exposure time increased, and (3) the rate of absorption increased as exposure concentration increased. Individual data were not provided.

Wahlberg and Skog (1963) used disappearance measurements of radiolabeled chromium to estimate dermal absorption of hexavalent chromium in vivo in guinea pigs. Animals were exposed for 5 hours to various concentrations (0.00048 - 4.870 molar) of sodium chromate labeled with ^{51}Cr . Dermal absorption of chromium was confirmed qualitatively by organ analysis. The maximal disappearance of hexavalent chromium was observed from a 0.261 molar solution. Of the 10 animals exposed to this concentration, the mean disappearance percentage per 5-hour period was 4% of the applied dose.

No studies could be located that examined dermal uptake of Cr(VI) from soils. However, chromium fate in soil and soil bioaccessibility studies (gastrointestinal and sweat leaching) have been conducted.

The relationship between Cr(VI) and Cr(III) in soil is a dynamic one, which is affected by soil type and mineral content, pH, solubility, and other factors (Bartlett, 1991; Fendorf, 1995; Stewart et al., 2003). Cr(VI) exhibits greater mobility and less adsorption in soils compared to Cr(III). Organic matter, Fe(II), and sulfides in soils are capable of reducing Cr(VI) to Cr(III), while manganese oxides in soils are capable of oxidizing Cr(III) to Cr(VI). Usually, part of any Cr(VI) added to soil will be reduced instantly, especially under acid conditions. However, high concentrations of polluting Cr(VI) may quickly exhaust the readily available reducing power of the matrix material and excess Cr(VI) may persist for years in soils without reduction.

Oral bioaccessibility of Cr(VI) from aged soils was determined by Stewart et al. (2003) using a physiologically based extraction test designed to simulate the digestive process of the stomach. It would be expected that bioaccessibility for dermal absorption of soil Cr(VI) would be no greater than oral absorption, and has been used to estimate dermal exposure to Cr(VI) in soil in previous health assessments (Sheehan et al., 1991).

In general, Cr(VI) bioaccessibility decreased with the aging of Cr(VI) in soils, with decreased bioaccessibility being most rapid for the first 50 days and then slowing dramatically between 50 and 200 days (Stewart et al., 2003). Chromium bioaccessibility was significantly influenced by reduction processes catalyzed by soil organic carbon. Soils with sufficient organic carbon had lower Cr(VI) bioaccessibility values of about 10 to 20% due to enhanced reduction of Cr(VI) to Cr(III). In soils where organic carbon was limited and reduction processes were minimal, considerably higher Cr(VI) bioaccessibility values of 60-70% were recorded.

Soil samples from two chromium waste sites that varied considerably in Cr(VI) concentration were extracted with a synthetic sweat solution to determine the potential for dermal bioaccessibility of Cr(VI) from contaminated soils (Wainman et al., 1994). The soils examined were contaminated with slag containing chromium from chromate and bichromate production facilities in New Jersey. One set of soil samples contained 710 µg Cr(VI)/g soil and contained chromate blooms, a thin layer of bright yellow crystals on the soil surface. Approximately 83% Cr(VI) was extracted in sweat from the soil with chromate blooms. Adjusting the pH of the soil from pH 5 to 8 had little effect on Cr(VI) extraction. In the other soil, the Cr(VI) concentration averaged 59 µg/g soil. Sweat extraction of Cr(VI) increased from 15 to 32% with increasing soil pH from pH 5 to 8. No Cr(VI) was extracted from the soil adjusted to pH 4. Extraction with distilled-deionized water was also performed, resulting in 76 and 27% extraction from soil with and without blooms, respectively.

Horowitz and Finley (1993) investigated the leaching of Cr(VI) in human sweat from chromite ore processing residue. The New Jersey ore residue originated from the same or similar processing facility as that investigated by Wainman et al. (1994). The human sweat at a pH of 7.2-8.0 extracted < 0.01% of Cr(VI) from the ore samples. Differences in the parent ore and extraction techniques were suspected to have led to the widely varying extraction of Cr(VI) from samples analyzed by Wainman et al. (1994) and Horowitz and Finley (1993).

Oral bioaccessibility studies have also been conducted on the New Jersey slag material (Hamel et al., 1999). Using two different methods, chromium in the slag material had an average bioaccessibility of 34 or 40%, depending on the method used.

F. 3.4.2 Discussion and Recommendation for a Hexavalent Chromium (Soluble Compounds) ABS

In the comprehensive in vitro study by Gammelgaard et al. (1992), a measurable increase in Cr(VI) penetrating full thickness human skin could not be detected with 48 hr exposure and only 1.1% of Cr(VI) had been absorbed into the skin. By 190 hrs of exposure fractional absorption of Cr(VI) increased considerably to 15%. The in vitro data indicates Cr(VI) salts have a long lag phase and are slowly absorbed. In contrast, the in vivo human study by Corbett et al. (1997) suggests a very short lag time for appearance of Cr(VI) systemically, with increased Cr levels in the circulatory system within 3 hrs of immersion in a water tank of dilute aqueous dichromate. The human in vivo study by Baranowska-Dutkiewicz, (1981) indirectly supports rapid dermal absorption of Cr(VI) with disappearance of aqueous Cr(VI) salt applied to skin for 15-60 min. Consequently, in vitro human exposure likely underestimates the dermal absorption potential of aqueous Cr(VI) solutions that occurs in vivo.

Alternatively, the indirect estimate of up to 23-44% dermal absorption of the applied dose of Cr(VI) salt by Baranowska-Dutkiewicz, (1981) and Mali et al. (1964) likely overestimates the dermal absorption potential due to use of a skin occlusion application and reliance on a disappearance method to estimate absorption. Mali et al. (1964) found only 0.35 µg of chromium in stratum corneum tape stripping even though a total of 22 µg of Cr(VI) was assumed absorbed by disappearance from the skin surface. This finding does not correspond with data by Liden and Lundberg, (1979) in which maximal levels of absorbed Cr(VI) was found in stratum corneum.

The 24 hr guinea pig in vivo study by Czernielewski et al. (1965) was the most comprehensive study available in regard to estimating whole body absorption of a dermally applied radiolabeled Cr(VI) solution. Analysis of excreta, blood, and most tissues yielded a fractional absorption of about 2.9%, of which 2.7% was found in excreta and blood. Dermal absorption in experimental animals often overestimates absorption in humans. The in vitro chromate disappearance constants for dermal exposures up to 24 hrs were 3-5 times greater through guinea pig skin compared to human skin (Wahlberg, 1965). However, recognizing that in vitro studies generate slower absorption rates of Cr(VI) than in vivo, the study by Czernielewski et al. (1965) provides a reasonable health protective absorption estimate (2.9%) when considering a human 48 hr in vitro fractional absorption of 1.1% was estimated by Gammelgaard et al. (1992).

To account for the effect of soil vehicle on dermal absorption of Cr(VI), the maximal Cr(VI) bioaccessibility of 83% in synthetic sweat as determined by Wainman et al. (1994) was taken into account. This bioaccessibility estimate was from a soil sample with about 710 µg Cr(VI) per g soil and contained chromate crystals on the soil surface. The contaminated soil probably represents a matrix described by Bartlett (1991) in which high concentrations of Cr(VI) exhausted the readily available reducing power of the soil and excess Cr(VI) persists on the soil surface without being reduced. Thus, multiplying 2.9% by 0.83 and rounded to the nearest whole number provides an ABS point estimate of 2% for Cr(VI) from soil vehicle.

The Hot Spots risk assessment procedures have previously assumed no reduction of deposited Cr(VI) because typically Cr(VI) deposition is modeled without soil sampling monitoring for the Cr(VI)/Cr(III) ratio and without an evaluation the redox potential of the soil. This assumption may result in overestimation of Cr(VI) soil concentrations in situations where Cr(VI) is readily reduced to Cr(III). Bioaccessibility is determined in part by the Cr(VI)/Cr(III) ratio. The use of soil with high concentrations of Cr(VI) to determine bioaccessibility is not likely to underestimate bioaccessibility under the conditions typically found in Hot Spots risk assessments, where Cr(VI) is deposited over a long period of time and typically results in lower soil concentrations than the 710 µg/g observed in the study by Wainman et al. (1994).

Limitations for the ABS not discussed above include lack of a factor for absorbed chromium lost through skin desquamation. Studies show that some Cr(VI) will be reduced to Cr(III) in skin and bind to cellular constituents (Gammelgaard *et al.*, 1992; Hostynek, 2003). If this occurs in the stratum corneum, the chromium will likely be removed through desquamation before systemic absorption can occur.

F.3.5 Fluoride and Soluble Fluoride Compounds

Recommended use of default inorganic compound ABS estimate of 1.0%.

F.3.5.1 Studies Considered

Excessive exposure to the negatively charged fluoride ion deposited on soil as an aerosol or as a soluble inorganic fluoride salt is known to have toxic effects in animals through ingestion of contaminated soil (Eagers, 1969). However, no quantitative data could be found regarding the fractional dermal absorption of soil-bound fluoride or fluoride compounds following contact with skin. Two animal studies observed elevated fluoride serum levels or systemic toxicity following dermal exposure to concentrated hydrofluoric acid, but immediate skin corrosion was apparent and likely influenced dermal absorption (Derelanko *et al.*, 1985; Boink *et al.*, 1995).

Much of the fluoride naturally present in soils or deposited from facility emissions will generally be in, or strongly adsorbed to, soil particles and is not in a form accessible for uptake by the body (Davison, 1987). Highest levels of water-soluble, or bioaccessible, fluoride in heavily contaminated soils was about 15-20% of total fluoride (Polomski *et al.*, 1982). Among several studies, the bioaccessible fluoride fraction in uncontaminated soils ranged from 0.06 to 7% of total soil fluoride (Gisiger, 1968; Polomski *et al.*, 1982; Milhaud *et al.*, 1989; Buykx *et al.*, 2004).

F.3.5.2 Discussion and Recommendation for a Fluoride and Soluble Fluoride Compound ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound fluoride, it is not possible to determine an ABS. Use of a 1% fractional absorption default value will likely not underestimate dermal absorption of soil-bound fluoride, given the highly ionic nature of fluoride and the strong adsorption of deposited fluoride to soil particles.

F. 3.6 Lead and Inorganic Lead Compounds

Recommended point estimate for dermal uptake: 3%

F. 3.6.1 Studies Considered

A. Key Study

The in vitro dermal absorption of lead oxide (PbO) powder ($<10\ \mu\text{m}$ particle diameter) in human abdominal skin was investigated (Filon et al., 2006). Each cell had a surface area of about $3.14\ \text{cm}^2$ and was filled with $5\ \text{mg PbO/cm}^2$ and with $2\ \text{ml}$ synthetic sweat at pH 5.0. At 24 hrs, a median of $2.9\ \text{ng/cm}^2$ (0.06% fractional absorption) had penetrated the skin to the receiving solution and a median of $321.3\ \text{ng/cm}^2$ (6.4% fractional absorption) was absorbed in the skin following surface decontamination. In another experiment, removal of PbO after 30 min exposure did not cause a reduction of Pb penetration in 24 hrs, but did cause a reduction in skin Pb content. This finding suggested that initial rapid absorption of Pb can occur during the first few min of exposure.

B. Supporting Studies

Bress and Bidanset (1991) studied percutaneous absorption of lead in vitro using human abdominal skin obtained from autopsy, and guinea pig dorsal skin. PbO or lead acetate ($10\ \text{mg}$) in saline solution was applied to $1.3\ \text{cm}^2$ skin samples. After 24 hours, the lead content of the saline reservoir fluid was measured. The lead content of the skin samples after exposure was not measured. In this experiment, 0.05% of the applied dose of lead acetate was recovered in the reservoir fluid, and less than 0.01% of the PbO. There was no difference between human and guinea pig skin.

Bress and Bidanset (1991) also examined in vivo percutaneous lead absorption in guinea pigs. Lead acetate or PbO, mixed in aqueous solution, was applied to a shaved area ($2\ \text{cm}^2$) of the back ($300\ \text{mg lead per kg body weight}$). After exposure for 1 week, the animals were killed and lead was measured in blood, brain, liver and kidney. Percent of applied dose absorbed could not be determined from this study. However, the concentration of lead in the measured tissues following lead oxide exposure was similar to that from control animals. In contrast, the lead concentration in measured tissues following lead acetate exposure was greater than controls, although absorption was considered poor, and statistics were not provided.

Moore et al. (1980) studied percutaneous absorption of lead acetate in humans from two commercial hair dye products. The products (one a lotion and one a cream) were spiked with lead-203 (^{203}Pb) and applied to each subject's forehead ($n=8$) for 12 hours. The preparations were applied in various forms (wet and dried) with periods of one month between each application. Lead absorption was estimated from blood counts, whole-body counts, and urine activity. Results were normalized for each subject by administration of an intravenous tracer dose of lead chloride.

The mean uptake of ^{203}Pb activity, measured in whole body at 12 hours, was greatest when the preparation was dried and skin was slightly abraded (0.18% of applied dose). The mean absorption including all methods of application (measured in whole body at 12 hours) was 0.058% with a range of 0-0.3%. It has been noted that the presence of colloidal sulphur in the lead acetate formulations used by Moore et al. (1980) may have led to the formation of insoluble lead sulfide, which would unlikely be significantly absorbed through skin (Stauber et al., 1994).

In a series of studies in human volunteers, aqueous solutions of inorganic lead salts including lead chloride and lead nitrate were shown to be rapidly absorbed through skin within 3-6 hrs and enter the extracellular compartment, resulting in increased concentrations of lead in the sweat and saliva but not the blood (Lilly et al., 1988; Stauber et al., 1994). However, application of radiolabeled lead (^{204}Pb) to skin of volunteers resulted in measurable increases of ^{204}Pb in the blood but with a very short residence time (Stauber et al., 1994). Preliminary experiments also showed rapid absorption of lead oxide and elemental lead through the human skin of volunteers and detected in the sweat within a few hours. Only PbCO_3 was not absorbed through skin. In mice, skin-absorbed lead concentrated more strongly in skin and muscle, and less in blood and other organs compared to intravenously injected lead (Florence et al., 1998).

The authors proposed that the behavior of skin-absorbed lead in the body is different from lead that is ingested or injected, in that lead which passed through skin is in a physicochemical form with low affinity for erythrocytes and a high affinity for extracellular fluid compartments. The implication is that testing blood for lead exposure may not fully account for absorption of lead through the skin.

Stauber et al. (1994) examined dermal lead absorption by placing lead nitrate and lead nitrate spiked with ^{204}Pb on the arms of volunteers for 24 hrs. Rapid increases of lead were observed in sweat samples from the unexposed arm and in saliva, but only small concentrations of lead in blood and urine. However, high levels of ^{204}Pb in blood and urine were measured 2 and 16 days, respectively, after exposure ended suggesting slow absorption of lead into the blood from lead retained in the skin.

In order to quantify dermal lead absorption, 4.4 mg lead (as 0.5 M $\text{Pb}(\text{NO}_3)_2$) was dispensed onto filter paper and secured with plastic wrap to the left arm of one subject. After 24 hours, the filter paper was removed and the arm was washed. Of the 4.4 mg lead, 3.1 mg was recovered from the filter paper and wash fluid. Using this disappearance technique, the authors estimated that 29% of the lead was absorbed into or through the skin. In two volunteers, the estimated excretion of skin-absorbed ^{204}Pb in the sweat of two volunteers over 24 hrs was 16 and 46 μg lead/L. Assuming an average sweat production of 500 ml/day, the authors estimated 0.6% and 1.5% of the total lead that was absorbed was excreted in sweat.

Lead acetate or nitrate was also applied to the skin of mice by the researchers in order to quantitate the amount of lead absorbed and retained in organs and tissues (Florence et al., 1998). Forty μl of aqueous solutions of the lead salts (6.4 mg of lead) was applied to a shaved area of skin and covered with Parafilm. Mice were sacrificed and organs and tissues analyzed for lead content after time periods of 2 hrs to 1 week. A total analysis of the organs, feces, and urine showed that, of the 6.4 mg of lead applied to the skin, 26 μg (0.4%) was absorbed through the skin and entered the circulatory system in 21 hrs. This analysis does not appear to include skin-absorbed lead at the site of application. No differences in absorption of the two lead salts were observed. Increased organ content of lead was noted by 6 hrs of exposure, with maximal organ concentrations generally occurring after 24-48 hrs of exposure.

To investigate the stratum corneum depth profiles of lead in lead battery workers, 10 repeated skin strips were collected from exposed skin (dorsal hand) and nonexposed skin (lower back) of 10 volunteers (Sun et al., 2002). Skin areas to be sampled were washed with soap and water, then ethanol, prior to collection in the morning before work. Total lead in stratum corneum strippings ranged from 20.74 to 86.53 μg (mean = 42.8 μg) from the hand, and 8.94 to 28.32 μg (mean = 17.4 μg) from the back. Approximately 20.8 μg (49%) of the total lead in the stratum corneum were in the first two tape strippings. There was a decreasing amount of lead content from both skin regions going from the outer to the inner layers, suggesting both regions had been contaminated with lead. Total amount of lead in the hand, but not the back, was linearly correlated with the amount of lead in blood. These findings indicate the source of lead in skin was from dermal exposure, rather than absorption of lead from the circulatory system into the skin.

Although the lead compound, which workers were exposed to, was not specified in the Sun et al. (2002) study, the primary lead compounds emitted during lead-acid battery production are identified as PbO and elemental lead (USEPA, 1998; Ruby et al., 1999). Elemental lead particles that are deposited in soils quickly form coatings of highly bioavailable PbO .

The leaching behavior of lead-contaminated soil can be divided into three stages based on the leachate pH: a high alkalinity leaching stage at $\text{pH} > 12$, where Pb formed soluble hydroxide anion complexes and leached out; a neutral to alkaline immobilization stage in the pH range of 6-12, which was characterized by low Pb leachability by adsorption and precipitation; and an acid leaching stage with $\text{pH} < 6$, where leachability increased exponentially with decreasing pH and was characterized as free Pb-ion (Jing et al., 2004). This study indicates that soluble Pb at the neutral pH found in most soils would only be a fraction of the total Pb content of the soil.

Several leaching studies of Pb-contaminated soils suggest the bioaccessible Pb in soil can vary greatly. Within a pH range of 7-8, soluble Pb ranged from less than 0.01% to 48% of total Pb content of soil (LaPerche et al., 1996; Yang et al.,

2001; 2002; Jing et al., 2004). In a major Pb contamination due to a paint spill the Pb soil content was 34,592 mg/kg, which is roughly an order of magnitude greater than many Pb-contaminated soils (Zhang et al., 1998). Soluble Pb at pH 7 was roughly estimated to be 18% of total soil Pb. At pH 5, fractional soluble Pb increased to about 41% of total soil Pb.

F. 3.6.2 Discussion and Recommendation for a Lead and Inorganic Lead Compound ABS

The accumulated in vivo absorption data did not provide enough quantitative information to estimate an ABS point estimate of lead including both systemic absorption and that retained in skin. Additionally, no data could be found that measured dermal absorption of lead from contaminated soil. Thus, the lead ABS point estimate incorporated data from an in vitro human study and soil leaching tests for lead-contaminated soil.

The most comprehensive human data available was the in vitro study by Filon et al. (2006), which observed 0.06% of applied lead penetrating to the receiving solution and 6.4% of applied lead retained in skin following dermal exposure of PbO in a synthetic sweat solution. The skin depth profile of lead shows 49% of the total lead in the stratum corneum was in the first two tape strippings, and might be removed through desquamation prior to systemic absorption (Sun et al., 2002). However, human in vivo dermal exposure data suggest a relatively short lag time for appearance of lead in blood and continual absorption of lead into the blood from the skin reservoir (Lilly et al., 1988; Stauber et al., 1994). Until further studies are conducted to estimate the fraction of lead removed via desquamation prior to systemic absorption, it is presumed that all the lead absorbed in skin is available for systemic absorption.

Although only 0.06% of the lead reached the receiving solution in the in vitro study by Filon et al. (2006), in vitro dermal absorption studies of metal salts generally do not include a full accounting of absorption due to skin shunts such as hair follicles and sweat ducts. Hostynek (2003) noted that these skin shunts swell shut upon hydration during in vitro dermal absorption studies, and can reduce the movement of some dermally applied metal salts directly into lower skin layers. The human in vivo data support the importance of sweat ducts for lead dermal absorption (Lilly et al., 1988; Stauber et al., 1994). In addition, the rapid reduction of lead dermal absorption early during exposure in the Filon et al. (2006) in vitro study has been considered evidence for skin shunts becoming hydrated and reducing lead absorption by these pathways (Hostynek, 2003). These data further support the reasoning that the lead retained in skin observed by Filon et al. (2006) cannot be discounted for potential systemic absorption.

In soil, aqueous leaching studies suggest soluble Pb can vary greatly depending on the soil characteristics. If sweat is the leachate, the pH can range between 4 and 7, with an average in male Caucasians of 4.85 (Wainman et al., 1994). The

acidic nature of sweat will likely enhance Pb bioaccessibility from soil compared to the soil pH ranges of 7-8. Because of the wide range of solubilities of Pb in soil, a health protective point estimate based on the solubility of a heavily Pb contaminated soil at a pH 5 (average pH of sweat) is warranted. Zhang et al. (1998) observed an approximate 41% Pb solubility at pH 5 from soil that may have been saturated with Pb paint (Pb content = 34,592 mg/kg soil). Adjusting the total fractional dermal absorption of 6.46% observed by Filon et al. (2006) by multiplying the fraction of soluble Pb in a highly impacted soil (0.41) determined by Zhang et al. (1998) results in an ABS point estimate of 3% after rounding to the nearest whole number.

A health protective ABS of 3% for Pb salts is higher than the other metal salts investigated. However, most of the soil leaching experiments used soils that were environmentally contaminated or incorporated time as a factor to control for soil aging. Absorption of Pb salts has also been shown to be high by the oral route relative to other metals, up to 90% absorption in the acidic environment of the stomach (Ruby et al., 1999).

F. 3.7 Inorganic Mercury Compounds

Recommended point estimate for dermal uptake from soil: 3%

F. 3.7.1 Studies Considered

Quantitative in vivo dermal absorption studies of Hg-contaminated soils have not been performed. A summary of the in vitro dermal studies exposing human and animal skin to Hg-contaminated soil are shown in Table F-2.

A. Key Studies

The dermal bioavailability of $^{203}\text{HgCl}_2$ was tested in vitro on dermatomed male pig skin as pure compound or following addition to sandy soil or clay soil (Skowronski et al., 2000). The Yorkshire pig model was chosen due to histological, physiological, biochemical and pharmacological similarities to human skin. The sandy and clay soil consisted of 4.4% and 1.6% organic matter, respectively, and a majority of the soil particles were in the range of 50-250 μm . A soil loading of 47 mg/cm^2 was calculated from the data provided and the HgCl_2 concentration was 5.3 ng/mg soil. Absorption was estimated up to 16 hrs following application.

In general, dermal absorption of Hg was greater from sandy soil than from clay soil. In both soils, the rate of appearance of Hg in the receptor fluid was rapid during the first hour, then decreased to a steady state for the remaining 15 hrs. In sandy soil freshly spiked with Hg, 0.28% and 37.5% of the applied dose had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. In clay soil freshly spiked with Hg, 0.08% and 39.7% of the applied dose

had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. For the pure compound, Skowronski et al. (2000) observed a skin penetration of 0.18%, but the amount bound to skin was 66.3%. For Hg aged 3 months in soil, dermal absorption was reduced to 3.3% in sandy soil and 2.6% in clay soil. Only 0.04% and 0.01% of these totals in the sandy and clay soil, respectively, represented percent of applied dose penetrating to the receptor fluid.

B. Supporting Studies

Radiolabeled mercuric chloride ($^{203}\text{HgCl}_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{203}\text{HgCl}_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 μm prior to spiking with the Hg salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2 . At 24 hrs, mean percent dermal absorption including the skin depot was 46.6 and 78.3% with and without soil, respectively. The fraction of total absorbed Hg that entered the diffusion cell in 24 hrs was 1.5 and 1.4% with and without soil, respectively.

A radiolabeled mercury compound ($^{203}\text{HgCl}_2$) was applied in soil or water vehicle to human skin in vitro (0.5 $\mu\text{g}/\text{cm}^2$ containing 1 μCi) for 24 hours (Wester et al., 1995; Wester and Maibach, 1998c). The investigators used Yolo County soil (26% sand, 26% clay, 48% silt, 0.9% organic) sieved for 180-300 μm particles. Receptor fluid accumulation from either water vehicle or soil vehicle was 0.07% of applied dose. Previously frozen or fresh skin gave similar results. Skin content of mercury from water vehicle averaged 29% of total dose applied. Using soil loads of 5, 10, and 40 mg, skin content of mercury was 10.4, 6.1, and 7.2% of dose applied, respectively.

In other human in vitro studies by the same research group, 5.5% absorption into skin and 0.01% penetration of pure HgCl_2 into receptor fluid was observed with a 30 min exposure (Wester et al., 1995; Wester and Maibach, 1998c). Continued perfusion for 48 hrs following the 30 min exposure increased skin absorption and penetration to receptor fluid to 6.3% and 0.09%, respectively, exhibiting the ability of Hg to migrate through skin after removal of Hg from the skin surface. When the in vitro exposure was increased from 30 min to 24 hrs, mercury skin absorption and penetration to receptor fluid was increased to 35.4% and 0.06%, respectively. No other results or methodology details were provided.

The dermal bioavailability of liquid and soil-bound $^{203}\text{HgCl}_2$ was tested on dermatomed human male skin in vitro (Sartorelli et al., 2003). For the liquid vehicle, HgCl_2 was added to buffered water solution (pH = 4.0). For the soil vehicle, HgCl_2 was added to loam soil consisting of 60% sand, 30% silt and 10% clay sieved to a particle size of <150 μm . Soil loading on skin was about 40

mg/cm², which would be greater than monolayer coverage using a particle size of <150 µm. The concentration of HgCl₂ was 0.0069 or 0.1190 nmol/cm³. After 72 hr exposure, any mercury absorbed from soil and penetrating skin to the receiving fluid was below the detection limit. Mean mercury concentrations in the skin were 10.53% of the applied low dose and 15.04% of the applied high dose. Mercury in the liquid vehicle was also applied at two concentrations of 0.0088 and 0.0607 nmol/cm³. At the low dose, percent of applied dose penetrating skin to the receptor fluid was 1.64 and 4.80% at 24 and 72 hrs, respectively. At the high dose, percent of applied dose penetrating skin to the receptor fluid was 0.34 and 0.93% at 24 and 72 hrs, respectively. Percent of applied dose retained in skin at 72 hrs was 18.93 and 44.97% for the low and high dose, respectively.

TABLE F.2. In Vitro Dermal Absorption Results of Mercuric Chloride from Soil

Study	Species	Exposure time (hr)	Soil fraction (µm)	% Reaching receptor	% Total absorbed fresh	% Total absorbed aged
Skowronski et al., 2000	pig	16	unsieved	0.28 ^a 0.08 ^b	37.8 ^a 39.8 ^b	3.3 ^a 2.5 ^b
Moody et al., 2009	human	24	90-710	1.5	46.6	ND ^c
Wester et al., 1995	human	24	180-300	0.07	7.9	ND
Sartorelli et al., 2003	human	72	<150	0 ^d	13	ND

^a Sandy soil

^b Clay soil

^c Not determined

^d Below the limit of detection

Hursh et al. (1989) studied dermal absorption of mercury vapor in humans. Each of 5 men exposed the skin of one forearm (a single exposure) to vapors with concentrations ranging from 0.88-2.14 ng ²⁰³Hg/cm³ for periods of 27 to 43 minutes. The rate of dermal uptake of mercury by the arm was quantified by measuring the difference between accumulated radioactivity on exposed and unexposed forearms following exposure. The mean uptake rate for the 5 subjects was reported as 0.024 ng Hg per cm² skin per minute per ng Hg per cm³ air. At this rate, the authors estimate that dermal absorption of mercury from vapor is approximately 2.6% of the rate of uptake by the lung.

In addition, the study protocol by Hursh et al. (1989) included a procedure in which adhesive strips were applied every 3-4 days post exposure for up to 40 days, which regularly removed cells of the stratum corneum from the same marked skin area following exposure. Larger amounts of Hg were stripped at later time points, suggesting that a substantial fraction of the absorbed Hg was probably associated or bound to keratinocytes rather than stratum corneum. Based on the whole body count of radiolabeled Hg and the amount of Hg

absorbed in the skin, the authors note that about half of the Hg eventually reached the bloodstream while the remainder was shed by desquamating cells. The data show estimates of 26, 43, 45, 45 and 46% of the dermally absorbed Hg reaching the bloodstream in the five volunteers. It was theorized that the elemental Hg penetrated the stratum corneum as vapor but that in the epidermis, some, but not all, of the Hg became oxidized to mercuric ions. The ions then became fixed or bound in the skin, some of which then moved upward and was eventually shed.

Baranowska-Dutkiewicz (1982) exposed the forearms of eight male volunteers to aqueous mercuric chloride solutions. Aliquots (0.25 ml) of HgCl_2 solutions were applied directly to a 22 cm^2 area of skin and covered with a watch-glass. Percutaneous absorption of mercury was calculated as the difference between the amount applied and the amount recovered after the skin and the watch-glass were washed. In order to examine the effect of concentration on uptake, 3 concentrations (0.01, 0.1, and 0.2 M) were applied for 30 minutes. As concentration increased, rate of uptake increased. In order to examine the influence of exposure time on uptake, 0.1 M HgCl_2 was applied for 5, 10, 15, 30 and 60 minutes. The authors reported that the average rate of uptake of mercury decreased from $9.3 \mu\text{g}/\text{cm}^2/\text{min}$ during a 5 minute exposure, to $2.5 \mu\text{g}/\text{cm}^2/\text{min}$ during a 1 hour exposure. The average percutaneous absorption of mercury was calculated for exposures of 5, 10, 15, 30, and 60 minutes resulting in 20%, 29%, 37%, 60% and 64% absorption of the applied dose, respectively.

In vivo application of aqueous HgCl_2 (0.1% w/v) to normal human skin followed by biopsy and visualization with electron microscopy found mercury deposits present intracellularly and extracellularly in the stratum corneum within minutes after application (Silberberg, 1972). The presence of mercury in the epidermis was not apparent until 2-4 hrs after application. The finding of immediate absorption of HgCl_2 correlates well with the in vivo findings of Baranowska-Dutkiewicz (1982), which observed the disappearance of HgCl_2 within 5 min after application to human skin.

An in vivo study in guinea pigs found that dermal absorption of Hg from HgCl_2 steadily decreases with increasing dose, suggesting a build up of a secondary diffusion barrier as a consequence of the electrophilic metal forming irreversible bonds with proteins of the skin (Friberg et al., 1961). Thereby a depot accumulates in the stratum corneum retarding further penetration in inverse proportion to metal concentration. This secondary barrier build-up retarding absorption was also evident with increasing dermal exposure intervals. HgCl_2 applied in vitro on human skin showed greatest percutaneous absorption during the first 5 hrs (Wahlberg, 1965). With later time periods the absorption rate decreased. The average absorption rate over the first 24 hrs was only about one-fourth the rate observed during the first 5 hrs of dermal exposure.

F. 3.7.2 Discussion and Recommendation for an Inorganic Mercury Compound ABS

More than 98% of mercury in soils is present as nonalkyl Hg(II) compounds and complexes, with direct deposition a significant component for much of the loading to terrestrial soils (Davis et al., 1997). In the soil, Hg can occur in three different valence states, namely as Hg^0 , Hg_2^{2+} and Hg^{2+} (Andersson, 1979). Hg^{2+} forms various complexes with OH^- and Cl^- ions, with the dominating mercuric complexes being HgCl_2 , $\text{Hg}(\text{OH})_2$ and HgOHCl . Only a small fraction of mercuric Hg species occurs free in solution; the major fraction is either bound to or in the soil material. Hg^{2+} and gaseous Hg^0 forms are preferably bound to organic matter in acidic soils, whereas in neutral and slightly alkaline soils, mineral components are active as well. Mercury exhibits a very high affinity for sulfide in reducing environments, forming relatively insoluble HgS (Davis et al., 1997).

Human skin both in vivo and in vitro has been shown to have a large capacity to accumulate metallic mercury vapor or mercury salts (as HgCl_2) applied in aqueous solution directly to skin. When freshly mixed with soil, Hg salts appear to have a greater ability for absorption into skin than other metal salts of concern in this section (i.e., Ni, Pb, Cd, etc.). However, similar to other metals, aging of Hg salt in soil significantly reduces the fractional absorption of Hg into skin. Therefore, a fractional absorption of 3% for HgCl_2 aged in soil prior to testing was chosen as the basis of the ABS to account for the aging affects in soil.

The Hg ABS is based on the in vitro study in pigs by Skowronski et al. (2000), in which HgCl_2 aged in soil for three months resulted in a considerable reduction of fractional absorption compared to HgCl_2 freshly mixed with soil. Limitations of this study include use of skin from a non-primate species, less than 24-hr exposure, and likely exceedance of soil monolayer coverage during the exposure. However, the human in vitro studies shown in Table F-2 also have their limitations for estimating fractional absorption, including exceedance of soil monolayer coverage (Sartorelli et al., 2003), or use of soil fractions that do not include soil particles less than 90 to 180 μm , which most commonly adhere to skin (Wester et al., 1995; Moody et al., 2009b).

Given the limitations, it is still unlikely that the ABS will underestimate fractional absorption. While both the human and animal in vitro studies show a large capacity for dermal absorption of Hg salt, very little reaches the diffusion cells (see Table F-2). Other studies reviewed here indicate that some of the Hg^{++} ions in mercuric salts tend to bind tightly to cellular proteins in all strata of skin, including stratum corneum, which may then impede further diffusion of mercury (Friberg et al., 1961; Silberberg, 1972; Hostynek, 2003). Mercury bound in stratum corneum would likely be removed via desquamation of skin. Hursh et al. (1989) has shown a considerable portion of absorbed Hg in skin will eventually be lost (up to 50%) due to desquamation.

Nevertheless, the development of a Hg ABS would benefit from human in vitro studies with Hg salts aged in soil, and continued monitoring after 24-hr dermal exposure to better estimate the amount of Hg that reaches the circulation (i.e., reaches the diffusion cells) and how much is likely to be lost due to desquamation. Because the ABS is based on Hg aged in soil, the ABS may underestimate fractional dermal absorption for soils in which a significant fraction of Hg has been very recently deposited on soil, or for soils that are heavily contaminated or saturated with Hg.

F. 3.8 Nickel and Nickel Compounds

Recommended point estimate for dermal uptake from soil: 4%

F. 3.8.1 Studies Considered

A. Key Studies

Radiolabeled nickel chloride ($^{63}\text{NiCl}_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{63}\text{NiCl}_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 μm prior to spiking with nickel salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2 . At 24 hrs, mean percent dermal absorption including the skin depot was 1 and 22.8% with and without soil, respectively. The fraction of total absorbed nickel that entered the diffusion cell in 24 hrs was 0.5 and 1.8% with and without soil, respectively.

In vivo, sequential adhesive tape stripping was implemented to characterize the penetration of nickel salt solutions in methanol and nickel metal powder in human stratum corneum following 24 hr occlusive application to the forearm (Hostynek et al., 2001a; Hostynek et al., 2001b). Hostynek et al. (2001a) investigated stratum corneum depth profiles for chloride, sulfate, nitrate and acetate nickel salts. Penetration of the stratum corneum by nickel salts at levels of 0.001-1% nickel salt was limited and closely related to the counter ion. The total percent dose of each salt recovered in stratum corneum was 26.1, 18.5, 8.8, and 3.3% for the nitrate, acetate, sulfate, and chloride, respectively. Tape stripping of the skin showed that most of the dose remains on the surface or was retained in the superficial layers of the stratum corneum. Depth profiles converged towards non-detectable levels in the lower stratum corneum regardless of concentration for the acetate, chloride and sulfate. Nickel applied as nitrate is retained at a constant level of approximately 1% of applied dose in the lower layers of the stratum corneum.

The in vitro permeation of 1% aqueous solutions of chloride, sulfate, nitrate, and acetate nickel salts across only the stratum corneum was investigated using

human leg skin (Tanojo et al., 2001). An initial surge in permeation rate within the first 24 hrs was observed for the nickel salts, followed by steady-state permeability rate up to 96 hrs that was not significantly different among the four salts. Nickel sulfate penetration of stratum corneum was greatest at 1.09%, whereas nickel nitrate recovery within stratum corneum was greatest at 0.95%. Total absorption (receptor fluid plus bound to stratum corneum) was 1.65, 1.49, 0.92, and 0.12 % for the sulfate, nitrate, chloride, and acetate salts, respectively. Total recovery of absorbed and unabsorbed nickel was virtually complete for all the salts except nickel nitrate, in which 84% recovery was attained.

Permeation of the salts was attributed by Tanojo et al. (2001) solely to the diffusion across the transcellular/intercellular barrier, as hair follicle and gland shunts were shut upon hydration by the aqueous solutions. These pathways swelling shut early during in vitro exposure may explain the decreased rate of absorption of nickel following an initial surge. Lack of ability to account for absorption of nickel via skin shunts may underestimate absorption.

B. Supporting Studies

Nickel reversibly binds to constituents of the epidermis when human epidermis was homogenized and incubated with nickel chloride solutions (Fullerton and Hoelgaard, 1988). Spruit et al. (1965) utilizing human cadaver skin has shown that nickel ions also reversibly bind to the dermis. Nickel powder has also been shown to oxidize when suspended in synthetic sweat, whereupon the metallic ions can be absorbed in vitro through human skin (Larese et al., 2007).

Under the same experimental exposure conditions as used by Hostynek et al., (2001a), nickel metal powder (particle size 3 μm) values were found to decrease from the superficial to the deeper layers of the stratum corneum (Hostynek et al., 2001b). However, nickel was still present at the deepest levels of stratum corneum removed by adhesive stripping, indicating that the metal has likely reached the viable epidermis and has potentially become systemically available. Although the data did not lend itself to estimation of a skin permeation rate, total nickel removed with 20 strips from the skin after 24 hr occlusion with 21.7 mg/cm^2 nickel powder was 38.7 $\mu\text{g}/\text{cm}^2$ (i.e., approximately 0.18% of the total nickel metal applied was found in the stratum corneum). These data indicated that in intact skin, nickel metal is oxidized to form soluble, stratum corneum-diffusible compounds which penetrate the intact stratum corneum.

Dermal absorption of nickel chloride as $^{63}\text{NiCl}_2$ from two different soils was determined in vitro through dermatomed pig skin cut 200 μm thick (Abdel-Rahman et al., 1997). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included $^{63}\text{NiCl}_2$ added immediately after the addition of the two soils (30 mg each) to skin, or after each soil was aged for 6 months with $^{63}\text{NiCl}_2$. Nickel chloride was also added alone in ethanol vehicle to separate skin samples. The chemical dose

was 113.8 ng/cm² and the soil loading was calculated to be 47 mg/cm². Monolayer coverage was probably exceeded with a soil loading of 47 mg/cm², causing a reduction in the observed fractional absorption.

Following 16 hrs of exposure, 0.3% of freshly applied ⁶³NiCl₂ in clay soil penetrated the skin to receptor fluid and 12.1% was found bound to skin. No significant difference for dermal absorption from sandy soil was observed. For the nickel solution applied to skin, 0.4 and 57.9% of the dose applied was found in receptor fluid and bound to skin, respectively. In aged sandy and clay soil, 0.03 and 0.05% nickel was found in the receptor fluid, respectively. Only 3.1 and 3.7% of the metal was bound to skin from sandy and clay soil, respectively. Aging nickel in the soils appeared to be complete by 3 months, as further aging in soil for 6 and 12 months did not result in further decreased dermal bioavailability of the metal (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

Fullerton et al. (1986) examined the permeation of nickel salts, specifically nickel sulfate and nickel chloride, through human full-thickness breast or leg skin in vitro. Skin excised in surgery was exposed to aqueous solutions of 184 µg/cm² for each nickel salt for up to 144 hrs. In the first experiment the effect of occlusion on the permeation rate of nickel chloride was examined. Occlusion resulted in a significantly higher permeation rate (approximately 3.6 percent of applied dose) compared with non-occluded exposure (approximately 0.23 percent) after 144 hours.

In the second experiment, nickel ions from a chloride solution was found to pass through the skin about 50 times faster than nickel ions from a sulfate solution. The amount of permeation of nickel chloride was much higher (16%) at 144 hours than nickel sulfate (0.3%). However, dermal penetration of the skin was slow, having a lag-time of about 50 hours. The occluded-skin permeation of nickel chloride was considerably higher in experiment 2 than experiment 1 (9-16% vs 3.6%) and was attributed by the authors to the use of breast skin from different donors.

In another study by the researchers, the stripping method was used in vitro on human full thickness skin following exposure to 5% nickel chloride in a 5% methyl cellulose gel for 96 hrs under occlusion (Fullerton et al., 1988). Nickel penetration from the gel solution gave similar results to nickel penetration of the pure nickel salt. Skin depth profiles found 50.9% was present on and in the stratum corneum (skin was not washed before stripping) with most of the nickel in the upper part of the stratum coeneum, 10.6% in the epidermis, 1.6% in the dermis, and only 0.4% reached the receptor solution.

Although the time frame and doses were different, similar dermal absorption results were obtained by Turkall et al. (2003) with in vitro dermal exposure of pig skin to 64 ng of radiolabeled nickel chloride. Penetration of ⁶³Ni in ethanol

through pig skin was 0.4% of initial dose and a total of 58% of the nickel remained in the skin at the end of 16 hrs.

F. 3.8.2 Discussion and Recommendation for a Nickel and Nickel Compound ABS

The only study that exposed human skin to soil contaminated with a nickel salt was the in vitro study by Moody et al. (Moody et al., 2009b). However, there is evidence to suggest in vitro tests for dermal absorption of nickel may underestimate absorption in vivo.

Hostynek et al. (2001a) observed a range of 26.1% to 3.3% absorption of applied dose over 24 hrs among four nickel salts tested in vivo on human stratum corneum. However, Tanojo et al. (2001) observed only a range of 1.65% to 0.12% absorption of applied dose over 96 hrs among the same four nickel salts tested in vitro on human stratum corneum. Comparison of these data indicates that reliance on in vitro absorption data probably underestimates the in vivo dermal absorption of nickel salts.

Specifically regarding the nickel chloride salt applied directly to skin, Hostynek et al. (2001a) observed a 24-hr total absorption of 3.3% for human skin in vivo, while Tanojo et al. (2001) observed a 96-hr total absorption of 0.92% for human skin in vitro. These data together suggests a 3.6-fold greater absorption in vivo compared to in vitro absorption.

Although the dermal absorption time used by Tanojo et al. (2001) was 96 hrs, most of the NiCl_2 had penetrated the skin in the first 24 hrs (probably greater than 95%) and appearance of nickel into the diffusion cells had attained steady state. Assuming steady state levels of NiCl_2 had also been reached in stratum corneum by 24 hrs, it can be estimated that the total absorption of NiCl_2 recorded by Tanojo et al. at 96 hrs was similar to that found at 24 hrs.

Applying a 3.6-fold in vivo/in vitro ratio adjustment to the fractional dermal absorption value of 1% for NiCl_2 determined by Moody et al. (2009b) results in an ABS value of 3.6% (or 4% when rounded to the nearest whole number). The ABS is similar to the fractional dermal absorption of 2-4% resulting from exposure of pig skin to NiCl_2 aged in different soils (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

F. 3.9 Selenium and Selenium Compounds

Recommended use of default inorganic compound ABS estimate of 1.0%.

F. 3.9.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption of soil-bound selenium (Se) or Se compounds applied to skin.

In dermal absorption studies of Se solutions, Farley et al. (1986) applied a 2.5% selenium sulfide lotion topically overnight on human volunteers. Skin region exposed and surface area covered were not described. Se levels in urine following exposure were significantly increased over control levels, but absorption was considered too slight to result in toxic effects. Repeated overnight treatments in a few volunteers over two days did not result in Se concentrations in the urine which were significantly higher than normal. In another study, increased serum levels of Se could not be measured in human volunteers that applied 2.5% selenium sulfide lotion to their torso overnight (Kalivas, 1993). Used in shampoo as a 1% selenium sulfide concentration, weekly use for a year did not change the normal urinary Se level (Cummins and Kimura, 1971).

Selenium sulfide is insoluble in water and is considerably less toxic via the oral route compared to elemental selenium or ionic forms of water-soluble selenite and selenate salts, such as sodium selenite (Cummins and Kimura, 1971). Lower gastrointestinal absorption of the sulfide salt was thought to be the cause of the lower oral toxicity.

The fraction of applied dose of ^{75}Se internally absorbed following application of selenous acid, a highly water soluble Se compound, onto the pelts of rats was calculated to be 1% per day over a 9-day exposure period (Medinsky et al., 1981).

F. 3.9.2 Discussion and Recommendation for a Selenium and Selenium Compounds ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound Se compounds, it is not possible to determine a chemical-specific point estimate ABS. However, use of a 1% fractional absorption default value for Se and Se salts for screening purposes will likely not underestimate dermal absorption of soil-bound Se, given that fractional absorption of highly soluble selenous acid applied neat to the pelts of rats was about 1% of applied dose.

F.4 Point Estimates for Dermal Absorption (ABS) of Organic Compounds

F. 4.1 Polychlorinated Biphenyls (PCBs)

Recommended point estimate for dermal uptake from soil: 14%

F. 4.1.1 Studies Considered

A. Key Study

The dermal uptake of each of the two commercial PCB formulations Aroclor 1242 and Aroclor 1254 was studied in vivo in female rhesus monkeys (Wester et al., 1993b). Aroclor 1242 is dominated by the tri- and tetra congeners (68 percent) and Aroclor 1254 is dominated by the penta- and hexa congeners (83 percent). Each PCB preparation was adsorbed onto soil particles that before sieving contained 26% sand, 26% clay, 48% silt, and 0.9% organic carbon. The soil was fractionated by particle size to 180 - 300 μm . The soil levels of the PCB preparations were 44 ppm Aroclor 1242 and 23 ppm Aroclor 1254.

The PCB laden soil was applied for 24 hours to a 12 cm^2 area of lightly shaved abdominal skin which was protected by a non-occluded patch. The applied doses were 1.75 $\mu\text{g}/\text{cm}^2$ Aroclor 1242 and 0.91 $\mu\text{g}/\text{cm}^2$ Aroclor 1254. The soil loadings were 40 mg soil/ cm^2 skin for both preparations. Following the first 24 hour exposure during which systemic absorption was measured as the content recovered in urine and feces, the patch was removed, the visible soil was removed from the site of application, the treated skin was washed with soap/water, and urine/feces were collected for an additional 34 days. One group of monkeys was exposed to the PCBs intravenously to adjust the cumulative urine/feces recovery of the dermally applied PCBs. The corrected fractional dermal absorption was 13.9% for Aroclor 1242 and 14.1% for Aroclor 1254.

B. Supporting Studies

PCBs are frequently found as complex mixtures of isomers in soil. To determine the effect of chlorine substitution on dermal absorption, Garner and Matthews (1998) applied dermal doses of ^{14}C -labeled mono-, di-, tetra-, and hexachlorobiphenyls to 1 cm^2 areas on the backs of rats for 48 hrs. Dermal penetration varied inversely with the degree of chlorination and ranged from essentially 100% for monochlorobiphenyl to about 30% for the hexachlorobiphenyl. However, the highly chlorinated PCBs tend to have slower metabolism and elimination and remain in the site of exposure longer, resulting in slow diffusion to the systemic circulation.

Mayes et al. (2002) dermally exposed female rhesus monkeys to radiolabeled Aroclor 1260 in soil in a manner similar to that used by Wester et al. (1993b). The soil was classified as sandy silt made up of 20% sand, 54% silt and 20% clay with a total organic carbon content of 5-6%. Sieving to <150 μm prior to application adjusted the total organic carbon content up to 8.7%. Five-hundred mg of soil either freshly spiked or aged for 88 days with PCBs (about 70 μg PCBs/g soil) was applied to a 12 cm^2 area of the chest/abdominal area and

protected by a non-occluded patch. The calculated dermal load was 42 mg/cm². One group was exposed to radiolabeled PCBs intravenously to adjust the cumulative urine/feces recovery of dermally applied PCBs. Groups exposed for 12 or 24 hrs to PCBs aged in soil exhibited percutaneous absorption values of 3.43 and 4.26%, respectively, while a group exposed for 24 hrs to soil freshly spiked with PCBs exhibited a dermal absorption value of 4.07%.

Mayes et al. (2002) stated that the reduction in fractional absorption compared to the Wester et al. (1993b) study was due to greater soil content of organic matter, which absorbs highly lipophilic compounds such as PCBs. However, the dermal load of 42 mg/cm² used by Mayes et al. likely exceeded monolayer coverage and caused a reduction in fractional absorption. No statistically significant difference was observed between the 12- and 24-hr exposure groups, suggesting PCBs partition quickly into lipid components of the stratum corneum. Likewise, aging of PCBs in soil had no effect on dermal absorption, suggesting rapid binding to the organic fraction of soil. The authors noted that Aroclor 1260 has a slightly higher octanol/water partition coefficient (log K_{ow}) than Aroclors 1242 and 1254 used by Wester et al. (1993b). A higher log K_{ow} would favor greater dermal absorption. However, the higher percentage of congeners with seven or more chlorines in Aroclor 1260 compared to Aroclors 1242 and 1254 tends to reduce dermal absorption, as shown by Garner and Matthews (1998).

The dermal absorption of radiolabeled 3,3',4,4'-tetrachlorobiphenyl (TCB) from liquid and soil mixtures was studied in an ex-vivo Yorkshire-Landrace pig-skin-flap model (Qiao and Riviere, 2000). The soil was described as a dust containing 31.2% sand, 16.8% silt, 53.0% clay (90% kaolinite) and 0.3% organic matter. No particle size fractionation was given. Sixty-five to 70 mg soil containing 200 µg of ¹⁴C-TCB (40 µg/cm²) was applied onto 5 cm² skin surface for 8 hrs, and the area was either left open (non-occlusive) or closed with Parafilm (occlusive). Greatest dermal absorption of TCB occurred from non-occluded soil. Fractional penetration of skin into the perfusate was 0.66%, absorption into dermis and other local tissues excluding stratum corneum was 2.48%, and stratum corneum absorption was 0.90%. Occlusion of the soil mixture significantly decreased dermal absorption 2-3-fold. In addition, dermal absorption from the liquid formulations (acetone, water-acetone mixture, or methylene chloride) was also significantly lower, suggesting TCB dermal absorption data from liquid formulations may considerably underestimate the risk of exposure to TCB in a soil matrix.

Qiao and Riviere (2001) performed a full mass balance in vivo study in Yorkshire-Landrace pigs after iv and dermal exposure to identical doses of 300 µg ¹⁴C-TCB. For dermal exposure, TCB in acetone vehicle was applied to a 7.5 cm² abdominal area of three pigs and protected by a glass chamber with holes, followed by covering with a nylon sieve screening. Urine and feces were collected for 11 days, with quantitative tissue analysis and tape stripping of the TCB-exposed dermal region conducted at the end of the 11 day exposure. On

average, about 70-71% of the applied dermal and iv doses were recovered. After iv dosing, a total of 60% of the dose was excreted via urinary and fecal routes with 8% of the initial dose remaining in body tissues. However, when TCB was given topically, the total excretion was only 5% but with a much larger tissue residue of 16%. The fraction of applied dermal dose reaching the systemic circulation was estimated at 22%, with 0.85% of the applied dose in stratum corneum following tape stripping of the TCB-exposed skin.

Because of the higher tissue residue levels following dermal absorption of TCB, the researchers noted that dermal absorption of chemicals similar to TCB may be underestimated without a full mass balance analysis (Qiao and Riviere, 2001). In other words, estimating dermal absorption by comparing urinary excretion or blood AUC data with data obtained by the iv route (which represents 100% absorption) would underestimate actual TCB dermal absorption. Use of these indirect methods of absorption would provide a calculated dermal absorption of 6.3-10%.

In addition to their in vivo monkey study described above, Wester et al. (1993b) also estimated in vitro dermal absorption of PCBs through human skin from soil. The percent dose penetrating to the receptor fluid after 24 hr exposure was 0.04% for both Aroclor 1242 and Aroclor 1254. The percent dose absorbed in skin was 2.6% for Aroclor 1242 and 1.6% for Aroclor 1254. The low in vitro dermal absorption compared to their in vivo monkey study results was thought to result from tissue viability issues or solubility limits with receptor fluid. However, in vitro dermal absorption and penetration using water as the vehicle resulted in a fractional absorption of 44-46% for both PCB formulations.

The dermal absorption of purified TCB from soil was studied in rat and human skin in vitro (USEPA, 1992). The soil was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. The TCB concentration in the soil was 1000 ppm and soil loading was 10 mg/cm² for the rat skin and 6 mg/cm² for the human skin. After 96 hours, 7.10% of the applied dose had penetrated the human skin into the perfusate, with another 0.26% remaining in skin after washing. In comparison, total dermal absorption in rat skin was over 4-fold higher. A similar experiment was conducted with rat skin in vitro using a soil with a high organic carbon content of 11.2%. Total dermal absorption of TCB was reduced over 3-fold compared to total absorption from the low organic carbon soil.

Dermal absorption of PCBs was estimated by the disappearance method in a single volunteer exposed to a mixture of ¹³C-labeled tetra-, penta-, hexa-, and heptachlorobiphenyls (Schmid et al., 1992). Five mg of the PCB mixture was applied to a 4 cm² cotton cloth in methylene chloride vehicle and dried. The cotton cloth was then applied to the tip of the forefinger or inner side of the forearm without occlusion for 8 hrs. After recovery of PCBs from the carrier and skin surface, disappearance of the remaining label suggested dermal absorption

was 7 and 47% of total dose applied to finger and forearm, respectively. However, plasma concentrations of ^{13}C -label were at or below the limit of detection (10-20 pg/ml) and were not considered reliable. Application of PCBs to aluminum foil, then rubbed into the skin of the forearm for 10 min resulted in a fractional absorption of 8% by the disappearance method and a plasma concentration of 56.3 pg/ml. The authors suggested that the lack of measurable serum levels of PCBs was partly due to evaporative loss during exposure.

Dermal absorption of HCB in vivo and in vitro was investigated in young (33 days of age) and adult (82 days of age) female rats (Fisher et al., 1989). Young rats absorbed 3.37 times as much HCB dermally as adults in the first 6 hrs of exposure. This resulted from a lag time for penetration of about 1 hr in young and 4 hrs in adult rats. At 72 hrs in vivo dermal penetration was 35% in young and 26% in adults compared to 1.5% for young and 1.0% for adult as measured with a continuous flow in vitro system, and 2.9% for young and 1.9% for adults as measured with a static in vitro system. By 120 hrs both young and adult rats have the same cumulative dermal absorption.

F. 4.1.2 Discussion and Recommendation for a Polychlorinated Biphenyl ABS

The Wester et al. (1993b) study provided the highest fractional dermal absorption value (14%) for PCBs in soil among the in vivo experimental animal species considered most relevant for human exposures (i.e., monkey and pigs). Similar to the Wester study, Mayes et al. (2002) used Rhesus monkeys to estimate dermal absorption of PCBs, but obtained fractional absorption values of only 3-4%. Suggested reasons for the lower value include a greater proportion of highly chlorinated congeners, which reduce absorption. However, this may not be an issue because Wester got similar fractional absorption values using an Arochlor (1242) dominated by tri- and tetra-congeners, and an Arochlor (1254) dominated by penta- and hexa-congeners. Use of a soil with higher organic carbon content may have also resulted in a lower fraction absorption. Additionally, Spalt et al. (2009) notes that Mayes et al. probably exceeded monolayer coverage during the experiment, whereas Wester et al. did not.

The Wester et al. and Mayes et al. studies also used an indirect mass balance adjustment for dermal absorption by comparing excretion of dermally-applied PCBs to excretion of iv administered PCBs. Qiao and Riviere (2001) showed that this may underestimate dermal absorption up to 2- to 3-fold due to greater organ and tissue content of PCBs following dermal absorption compared to PCBs that were injected by the iv route. Thus, the highest absorption fraction estimate (14%) by Wester et al. (1993b) is recommended as the best health protective value.

Wester et al. (1993b) did not age the PCBs in soil prior to dermal application on the monkeys. However, Mayes et al. (2002) observed that aging of PCBs in soil did not reduce dermal absorption compared to freshly spiked soil.

In vitro dermal absorption studies were not considered for estimating the ABS. Comparison studies applying PCBs both in vivo and in vitro suggests that estimating dermal fractional absorption with an in vitro system would underestimate dermal absorption obtained by in vivo methods (USEPA, 1992; Wester et al., 1993b). A reason for this underestimation may be the limited lipophilicity of the receptor fluid used with the in vitro systems.

F. 4.2 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans

"Dioxin" emissions are reported as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) equivalents. Therefore, for purposes of the Hot Spots program, all polychlorinated dibenzo-p-dioxins and dibenzofurans are considered to have the same dermal absorption characteristics as TCDD.

Recommended point estimate for dermal uptake from soil: 3%

F. 4.2.1 Studies Considered

A. Key Studies

The dermal absorption of TCDD from high organic (HOS) and low organic (LOS) soils in rats in vitro, and in human skin in vitro and rats in vivo from LOS only, was investigated during exposure intervals up to 96 hours (U.S. EPA, 1992; Roy et al., 2008). The LOS was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. For the in vitro studies, the TCDD concentration in the LOS was 1 ppm with soil loading of 10 mg/cm² on the rat skin and 6 mg/cm² on the human skin. After 24 hrs, 0.28% and 1.17% of the applied dose had penetrated human and rat skin, respectively, to the receptor fluid (Table F-3). Although the dose of TCDD remaining in skin was not determined at 24 hrs, the 96 hr exposure estimate in human and rat skin following skin surface wiping was 0.17 and 1.41%, respectively. The percent of applied dose reaching the receptor fluid at 96 hrs was 2.25% in human skin and 6.32% in rat skin.

The percent of dose absorbed from LOS by rats in vivo was 7.9% at 24 hrs and 16.3% at 96 hrs (Table F-3). TCDD absorbed was estimated indirectly by dividing the percent of applied dose found in the excreta by the fraction of applied dose in the excreta at the same time after i.v. administration. However, TCDD systemically absorbed at 96 hrs was also quantified in all urine, feces and tissues, resulting in 16.3% of dose absorbed. To derive an ABS for human *in vivo* uptake of TCDD from LOS and HOS (11.2% organic carbon content), USEPA (1992) applied corrections by direct ratios to account for rat in vivo, rat in vitro, and human in vitro data. For human TCDD absorption from LOS, the in vivo absorption in rat at 24 hrs was multiplied by the ratio of human to rat total absorption in vitro measured at 96 hrs. The 96 hrs data were used because this

was the only measurement in which TCDD in skin was quantified. The final ABS was 2.5% ($8.0\% \times 2.42\% / 7.74\%$).

Table F.3. Percent Dermal Absorption of TCDD over Time from Low Organic Soil^a

Time (hr)	Rat – in vivo	Rat – in vitro	Human in vitro
24	7.9	1.17	0.28
96	16.3	6.32	2.25
96 (Dose in skin sample after wiping)	NA ^b	1.4	0.2
96 (Total)	16.3	7.7	2.4

^a Data from US EPA (1992) and Roy et al., 2008

^b Not applicable

Roy et al. (2008) note that steady state conditions for the TCDD concentration in skin from LOS are reached by 24 hours for the in vitro experiments. Thus it should be reasonable to assume that the amount in the skin after 96 hours is about the same as after 24 hours. The researchers also observed that the rat in vivo percent absorbed results were about twice as high as the rat in vitro results after 96 hours. Assuming the human in vitro results would operate in a similar fashion Roy et al. obtained a human 24-hr fractional TCDD absorption rate of 0.96% ($0.48\% \times 16.3\% / 7.7\%$). Additionally, a fractional absorption value of 0.1% was derived for TCDD absorbed from HOS (soil with an organic content >10%).

Alternately, it may be more relevant to multiply the rat in vivo percent absorbed at 24 hours (7.9%) by the estimated in vitro rat-to-human ratio for total percent TCDD absorbed at 24 hours ($0.48\% / 2.75\%$), rather than rely on any of the results from 96 hr exposure. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%.

B. Supporting Studies

Shu et al. (1988) applied soil-bound TCDD to the backs of rats, clipped of hair. Laboratory contaminated TCDD soil was prepared from soil obtained from Times Beach MO and determined not to contain TCDD before the experimental addition of the chemical. Environmentally contaminated soil was also obtained from Times Beach, MO and determined to contain 123 ppb TCDD after sieving through a 40-mesh screen. The organic carbon content of the soils was not specified. Soil loading was 20.8 mg soil/cm² skin on a total skin area of 12 cm². The TCDD content of the laboratory prepared soil was 10 or 100 pg/mg soil. Occlusion of the skin was minimized by the use of a perforated aluminum eye patch to cover the exposed area. Dermal exposure duration to the TCDD-laden soil was 24 hours and recovery was measured 48 hours following initiation of

exposure. In some experiments, 0.5 or 2.0 percent (w/w) used crankcase oil was added to the soil before the addition of TCDD.

Following 24 hour dermal exposure + 24 hour post-exposure (total of 48 hours from initiation of exposure), the TCDD content of the liver was determined. The uptake of TCDD under the experimental protocols ranged from 0.54 ± 0.06 to $1.01 \pm 0.22\%$ and averaged $0.76 \pm 0.16\%$. The percent uptake of TCDD in liver was not affected by either the applied TCDD dose (12.5 or 125 ng/kg BW), the presence of crankcase oil in the soil, the use of soil that had been environmentally contaminated with TCDD, or by the use of haired or hairless rats.

Peak liver concentrations for TCDD administered orally and dermally were used to correct for incomplete absorption in the calculation of relative dermal absorption. The calculation is based on the assumption that the source of fecal TCDD following oral exposure is unabsorbed TCDD. The estimated relative dermal bioavailability is 1.5% from laboratory-contaminated soil and 1.6% from environmentally contaminated soil.

Diliberto et al. (1996) note that during the first 48 hours following oral exposure, TCDD in rat feces included both unabsorbed TCDD and absorbed TCDD that was excreted in bile. However the data suggest that at 48 hours, absorbed TCDD contributes only about 10% of the fecal TCDD.

Poiger and Schlatter (1980) applied radiolabeled TCDD in a soil/water paste formulation (26, 350, or 1300 ng in 14.3 mg soil/cm² skin) to the backs of hairless rats and measured the appearance of label in the livers. The soil (organic carbon content unspecified) was taken from the Seveso region and was TCDD-free. Measurements were taken 48 hours after the initiation of a 24 hour exposure period.

The average percentage of dose in the liver after dermal application was 0.05, 1.7, and 2.2% for the 26, 350, and 1300 ng dose groups, respectively. The authors noted that other researchers observed that 70% of total body burden of administered TCDD is found in the liver of rats. Using this estimate, the corrected dermal absorption of total applied dose is 0.07, 2.4, and 3.1% for the 26, 350, and 1300 ng dose groups, respectively. The authors also compared the liver uptake of dermally applied TCDD from a soil/water paste to the uptake from methanol, and found the soil/water paste caused a reduction in the fractional uptake (compared to methanol) of 12 percent (1.6 ng TCDD/kg BW) or 15 percent (5.8 ng/kg BW).

TCDD in acetone vehicle was applied to human skin in vitro to estimate the capacity of skin to store TCDD (Weber et al., 1991). Although TCDD did not readily penetrate the skin into the saline receptor fluid (0.03% of dose) after 16.7 hrs exposure, a major portion of the dose was found in skin. The percent of dose

absorbed in skin at 16.7 hrs was 56% at a skin loading of 65 ng/cm², and 40% at a skin loading of 6.5 ng/cm².

Age may be a factor in the absorption of TCDD-like compounds. Anderson et al. (1993) applied radiolabeled TCDD in acetone (111 pmol/cm² applied over 1.8 cm²) to the interscapular region of 3-, 5-, 8-, 10-, and 36-week-old rats and measured dermal absorption 72 hrs later. Dermal absorption was greatest in 3-week-old rats at 64%, decreasing to about 40% in 5-, 8-, and 10-week-old rats, and to about 22% in 36-week-old rats. Although the reason for the age-related changes in dermal absorption was not explored, the authors suggested increased lipids in skin of the young may be a factor.

F. 4.2.2 Discussion and Recommendation for a Polychlorinated Dibenzo-p-dioxin and Dibenzofuran ABS

Human skin has the capacity to store TCDD in vitro (Weber et al., 1991; Roy et al., 2008). Once absorbed in skin, lipophilic compounds such as TCDD are anticipated to be eventually absorbed into the systemic circulation. Data for another lipophilic pollutant, lindane, indicates that the chemical retained in skin will be eventually systemically absorbed (Dick et al., 1997a).

Several methods for assessing the dermal exposure data by US EPA (1992) and Roy et al. (2008) were employed above to obtain a total fractional absorption (i.e., amount that reached the bloodstream + amount retained in skin) for TCDD ABS. Since the fractional dermal absorption values presented in this document are based on 24-hr exposure, the most relevant means for estimating an ABS is to rely only on the 24-hr absorption results. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%. Roy et al. (2008) employ a monolayer adjustment factor in their assessment, noting that the human in vitro skin test used a soil load of 6 mg/cm², which was greater than monolayer load by a factor of 2. Multiplying by this factor, the 24-hr TCDD fractional absorption for human skin is estimated at 2.8% for LOS, which is then rounded up to 3%.

Although both Shu et al. (1988) and Poiger and Schlatter (1980) estimated dermal absorption fractions in rats near 2%, neither study specified the organic carbon content of the TCDD-contaminated soil. The organic carbon content of soil is a major determinant for TCDD dermal absorption. At 96 hrs, USEPA (1992) noted that the ratio of TCDD absorption from low organic carbon soil (0.45% organic carbon) in rat skin measured in vitro to absorption from high organic carbon soil (11.2% organic carbon) in the same system was 7.5. Without the organic carbon content of the soil, it is difficult to compare the findings of Shu et al. (1988) and Poiger and Schlatter (1980) with that of the USEPA study.

TCDD aged in soil prior to dermal application had little effect on absorption, which is supported by the long half-life of TCDD in soil. Shu et al. (1988) observed similar dermal absorption estimates when TCDD was freshly added to

soil in the lab and soil that had been environmentally contaminated with TCDD and presumably aged in the soil. In addition, soil aging of polychlorinated biphenyls (PCBs), a group of soil contaminants with some structural similarities to TCDD, is not a significant factor for dermal absorption (Mayes et al., 2002). On the other hand, oral studies of soil-laden TCDD do indicate aging to be a factor in the reduction of TCDD intestinal absorption (Poiger and Schlatter, 1980).

F. 4.3 Polycyclic Aromatic Hydrocarbons as Benzo[a]pyrene (BaP)

Recommended point estimate for dermal uptake from soil: 13%

Field studies of workers have shown that dermal absorption of PAHs may be significant. Dermal absorption of PAHs, based on the urinary excretion of 1-hydroxypyrene (1-HP), has been documented among petrochemical industry workers, including those digging in PAH-contaminated soil (Boogaard and van Sittert, 1995). Although no attempt was made to quantify the extent of absorption through dermal and inhalation routes, the results of the study strongly suggest dermal uptake is substantial and is mitigated by the use of appropriate protective clothing. Elovaara et al. (1995) compared the levels of urinary 1-HP among 6 creosote workers compared to that expected from the inhalation of the known air levels of PAHs containing ≥ 4 rings. Higher levels of urinary 1-HP were observed than could be accounted for solely from the inhalation route of exposure.

F. 4.3.1 Studies Considered

A. Key Study

In Wester et al. (1990b), the dermal uptake of soil-bound BaP was studied in vivo in four rhesus monkeys. The systemic absorption of soil-bound BaP was based on urinary excretion following exposure of 12 cm² abdominal skin to 10 ppm BaP in soil at a soil loading of 40 mg/cm² skin. A nonocclusive cover protected the dermal application site. Prior to sieving to approximately 180-320 μ m diameter, the soil composition was 26 percent sand, 26 percent clay, and 48 percent silt with 0.9 percent organic carbon content.

Exposure duration to the chemical laden soil was 24 hours, during which time urine was collected. The cover was removed, visible soil was collected, and the skin application site was washed with soap and water. Urine was then collected for 6 additional days for a cumulative recovery period of 7 days. Incomplete excretion of BaP was corrected by the urinary excretion of BaP following intravenous (iv) administration of the PAH in acetone. The authors report a mean 24 hour dermal absorption factor of 13.2 ± 3.4 percent (Table F.4).

Radiolabeled BaP (¹⁴C-BaP) was mixed with commercial gardening soil and applied in vitro onto fresh human female breast skin (obtained within 1 day of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2007).

The same amount of ^{14}C -BaP was also applied without soil to human skin samples. The soil had been sieved to $<710\ \mu\text{m}$ prior to spiking with BaP. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of $5\ \text{mg}/\text{cm}^2$. At 24 hrs, the mean total percent dermal absorption including the skin depot was 14.8 and 56.4% with and without soil, respectively. The fraction of total absorbed BaP that entered the diffusion cell in 24 hrs was 7.2 and 11% with and without soil, respectively.

B. Supporting Studies

Yang, et al. (1989) studied the in vivo systemic absorption in rats of BaP in soil, fortified with petroleum crude oil (1 percent (w/w)) to which ^3H -BaP was added. The soil, which consisted of 46 percent sand, 18 percent clay and 36 percent silt, with an organic content of 1.6 percent, was sieved to a particle size $<150\ \mu\text{m}$. The final BaP level in the soil was 1 ppm and the soil loading was $9\ \text{mg}/\text{cm}^2$.

After 24 hours, 1.1 percent of the radioactive label was found in the rat urine and feces; no label was found in the tissues. By 96 hours (4 days) the cumulative total of radioactive label in the excreta + tissues was 9.2 percent, of which 5.8 percent was in the feces. The dermal uptake rate was estimated to be $0.2\ \text{ng}/\text{cm}^2/\text{day}$. Remaining BaP retained in skin at the site of application was not determined. In vitro absorption of BaP in soil was also determined in rats using a similar exposure protocol. Very good correlation was observed between the in vivo and in vitro data.

In conjunction with the in vivo dermal absorption studies in monkeys, Wester et al. (1990b) also conducted BaP dermal absorption experiments with viable human skin in vitro. Under the same soil and loading conditions of the in vivo monkey study, BaP-laden soil was applied to skin samples (dermatomed to $500\ \mu\text{m}$ thickness) for 24 hrs. The percentage of applied dose in skin and in human plasma receptor fluid was 1.4 and 0.01%, respectively. When acetone was used as the vehicle under the same exposure conditions, BaP found in receptor fluid and in skin was 0.09 and 23.7% of applied dose, respectively.

Dermal absorption of ^3H -BaP from two different soils was determined in vitro through dermatomed pig skin cut $200\ \mu\text{m}$ thick (Abdel-Rahman et al., 2002). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included: BaP applied as the pure compound; BaP applied immediately after the addition to each soil type (30 mg each); and pre-sterilized soils aged for three months with BaP. The chemical dose was $1.67\ \text{mg}/\text{kg}$ and the soil loading was calculated to be $47\ \text{mg}/\text{cm}^2$.

Following 16 hrs of exposure, 0.2% of freshly applied BaP in sandy soil penetrated the skin to receptor fluid and 8.3% was found bound to skin. In clay soil, 0.1% of freshly applied BaP was found in the receptor fluid and 3.3% was bound to skin. In comparison, pure BaP applied to skin resulted in 0.2 and

75.8% of the dose found in receptor fluid and bound to skin, respectively. For BaP aged in either sandy or clay soil, 0.1% was found in the receptor fluid. Only 3.7 and 1.7% were bound to skin from sandy and clay soil, respectively. Aging BaP in the soils for three months decreased total dermal adsorption by about 2-fold compared to BaP freshly applied to the soils.

Table F.4. In Vivo and In Vitro Dermal Absorption Results of Pure BaP Freshly Applied or Aged in Soils

Study	Species Treatment	Exposure time (hr)	Soil fraction (µm)	% Total absorbed fresh	% Total absorbed aged
Wester et al. 1990b	monkey in vivo	24	180-320	13.2	ND ^a
Yang et al., 1989	rat in vivo	96	<150	9.2	ND
Moody et al., 2007	human in vitro	24	<710	14.8	ND ^c
Wester et al., 1990b	human in vitro	24	180-320	1.4	ND
Abdel-Rahman et al., 2002	pig in vitro	16	unsieved	8.5 ^b 3.4 ^c	3.8 ^b 1.8 ^c

^a Not determined

^b Sandy soil

^c Clay soil

Studies were conducted to measure in vitro absorption of BaP through human skin (previously stored frozen) from contaminated soils at manufactured gas plant (MPG) sites. These sites were impacted by PAHs in lampblack, a residue produced from the pyrolysis of oil to produce gas. Roy et al. (1998) collected nine soils from three MPG sites containing targeted PAHs at levels ranging from 10 to 2400 mg/kg. Dermal penetration rates of target PAH from the soils were determined using ³H-BaP as a surrogate. Soils were sieved to <150 µm prior to analytical characterization and loaded onto skin sections at 25 mg/cm². Dermal absorption tests ran up to 144 hrs. The recovery of radiolabel in the receptor fluid ranged from 0.19 to 1.0%, while radiolabel absorbed in skin ranged from 0.4 to 1.0%. The highest percent of applied dose (receptor fluid + skin) from a contaminated soil was 1.9%.

Contaminated soils were collected from 7 oil-gas MPG sites in California to assess dermal absorption of BaP in vitro (Stroo et al., 2005a; Stroo et al., 2005b). The soil was sieved to <150 µm and loaded onto human skin at 10 mg/cm². The skin samples were dermatomed to a thickness of 350 µm. The percentage of applied dose absorbed across skin over 24 hrs ranged from 0.14 to 1.05%. The lower absorption of BaP in the lampblack samples compared to the Wester et al. (1990b) study was attributed to soil aging effects, but also to

tighter binding of BaP to lampblack. Lampblack tends to bind hydrocarbons more tightly than conventional soil organic matter.

To investigate effects of soil loading and aging on PAH dermal absorption, Roy and Singh (2001) loaded PAH-spiked soil onto human skin sections at 1, 2.5, 5 and 10 mg/cm² following aging of the PAHs in soil up to 110 days. A field soil was sieved to <150 µm, resulting in a total organic content of 0.43%. The soil was spiked with coal tar and ³H-BaP to achieve a final soil BaP concentration of 65 ppm. At soil loadings of 1 and 2.5 mg/cm², approximately 1% of the applied dose was in the receptor fluid at 24 hrs. The percent of applied dose absorbed decreased with increasing soil loadings of 5 and 10 mg/cm², respectively, indicating skin loading above monolayer coverage. In the aging experiment, the dermal bioavailability of coal-tar-derived BaP was reduced by about half by day 110 compared to the soil freshly spiked with ³H-BaP.

The in vitro dermal absorption of BaP applied in acetone to full-thickness skin was compared among six mammalian species (Kao et al., 1985). The percent of applied dose permeating fresh, viable skin in 24 hrs was approximately 10% in mice, 3% in marmosets and humans, 2% in rats and rabbits, and <1% in guinea pigs. However, permeation through skin rendered non-viable by previous freezing was <1% of applied dose in all species. Permeation was accompanied by extensive first-pass metabolism of BaP in viable skin of all species. Nearly half the BaP that permeated viable human skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid.

PAHs have been shown to be poorly absorbed through skin from solids. No percutaneous penetration of PAHs from coal dust occurred across human skin in vitro (Sartorelli et al., 2001).

F. 4.3.2 Discussion and Recommendation for a Polycyclic Aromatic Hydrocarbon ABS

A fractional dermal absorption of 13% determined in a primate species in vivo represents a health-protective estimate of human systemic absorption of pure BaP freshly applied to an agricultural soil (Wester et al., 1990b). In support, a similar in vitro fractional absorption (14.8%) was attained by Moody et al. (2007) for 24-hr exposure of human skin to BaP-contaminated soil. The work by Wester et al. and Moody et al. were also one of the few BaP exposure studies that did not exceed monolayer soil coverage of the skin, although the coarse particle soil loadings used in the monkey study may have resulted in a lower fractional absorption.

The only other in vivo study of BaP dermal absorption from soil was in rats, in which a lower fractional absorption of 9.2% was estimated after 4-day exposure (Yang et al., 1989). Although higher organic content of the soil used could be a

factor in the lower ABS in rats, the presence of petroleum crude oil (1 percent (w/w)) as a co-contaminant was also likely a factor in the lower absorption in rats compared to monkeys. Stroo et al. (2005a) note that tar in contaminated soils tends to bind hydrocarbons more tightly than conventional soil organic matter and reduces bioavailability for dermal absorption. In addition, a soil loading of 9 mg/cm² exceeds monolayer coverage with soil sieved to <150 µm causing a further reduction in the percent fractional absorption.

Wester et al., (1990b) observed a roughly 10-fold lower fractional absorption of BaP in human skin in vitro compared to the human in vitro study by Moody et al., (2007). Use of a coarse soil fraction (180-320 µm) by Wester et al. may have reduced dermal absorption. The reduction in absorption may also be due, in part, to loss of skin viability. The Wester study used cadaver skin up to 5 days after harvest. The studies of Moody et al. obtained human skin in as little as 2-24 hrs after live donor skin harvest.

The metabolic viability of the skin samples used for in vitro studies is a factor that can affect skin permeation of BaP. Kao et al. (1985) have shown that the rate of cutaneous metabolism of BaP has a positive correlation with the permeation rate of BaP through viable skin. For example, using previously frozen human skin, as was done in some studies discussed above, renders the samples less viable and possibly much less permeable to BaP. When BaP was applied in vitro to fresh skin samples and previously frozen skin from the same individuals, a significant reduction in dermal absorption into the receiver solution was observed for the previously frozen skin (Moody et al., 2009a). However, when the skin depot was included, the difference in dermal absorption between fresh and previously frozen skin was not as pronounced.

The dermal exposure algorithm presented in Chapter 6 includes a half-life variable for BaP in soil, although it is generally assumed the half-life reflects primarily the loss of chemical due to microbial degradation. However, Abdel-Rahman et al. (2002) showed that aging of BaP in sterile soil also resulted in decreased fractional absorption in pig skin. This finding suggests BaP also shows reduced bioaccessibility over time due to partitioning into more remote sites within the soil matrix. Vigorous soil extraction procedures often used to assess soil half-life may overestimate the bioavailability of BaP because it may not be a true representation of BaP's bioaccessibility in soil for dermal absorption. Extraction techniques using human sweat or synthetic sweat would provide a more accurate estimate of the BaP half-life in soil for fractional dermal absorption studies.

F. 4.4 *Hexachlorobenzene*

Recommended use of default organic compound ABS estimate of 4%

F. 4.4.1 Studies Considered

No experimental data are available investigating the dermal absorption of HCB from contaminated soil. In a rat in vivo study, ^{14}C -HCB dissolved in tetrachloroethylene was applied neat to the skin and covered with an occlusive patch after the vehicle had evaporated (Koizumi, 1991). The cumulative mean absorbed body burden, not including dosed skin directly contaminated, was 2.67% after 24 hours. Approximately 5% of the total dose remained in or on the dosed area of skin prior to washing. Washing the dosed area of skin resulted in removal of 4% of the total dose, indicating that 1% of the total dose was absorbed in the skin on which ^{14}C -HCB was directly applied.

A Monte Carlo simulation was developed to produce a probability density function for the dermal uptake fraction of HCB in soil deposited on human skin (McKone, 1991). A two-layer model was used that accounted for chemical properties, skin properties, soil properties, and exposure conditions. The resulting modeled daily dermal uptake fraction had an arithmetic mean value of 0.15 per day (24 hrs), and an arithmetic standard deviation of 0.18 per day.

F.4.4.2 Discussion and Recommendation for a Hexachlorobenzene Compound ABS

A single dermal absorption study in rats observed a 24-hr fractional absorption of 4% (rounded to nearest whole number) for the neat compound. This estimate includes HCB retained in skin at the site of application. Absorption of HCB may have increased as a result of occlusion of the exposed skin area to prevent evaporation of HCB.

A default ABS of 4% is recommended based on the rat dermal exposure study, although the chemical was applied neat to the skin. The HCB modeling study by suggests that the fractional absorption of HCB in soil may be 15%, so no adjustment was made to the ABS to account for reduced absorption due to partitioning to soil organic matter (McKone, 1991). In support, HCB is structurally similar to hexachlorocyclohexane (HCH), which has an ABS of 3%. However, the K_{ow} for HCB ($\log K_{ow}$ 5.73) is about 100 times greater than that of the HCHs, which would suggest a greater ability for absorption into skin. On the other hand, the high K_{ow} also indicates that HCB will have stronger sorption to soil organic material compared to the HCHs, which usually decreases the dermal absorption potential. Until more relevant dermal absorption studies are conducted, an ABS of 4% is recommended for HCB.

F. 4.5 Hexachlorocyclohexanes

Hexachlorocyclohexanes (HCHs) occur as eight isomers. The most common isomer is the gamma, which when purified to 99%, was sold under the trade name of lindane. Lindane was a widely used pesticide but almost all uses of

lindane have been banned in the United States due to carcinogenicity concerns, high biopersistence and bioaccumulation. Dermal absorption data exist only for lindane, thus all HCH isomers are considered to have the same dermal absorption characteristics as lindane.

Recommended point estimate for dermal uptake from soil: 3%

F. 4.5.1 Studies Considered

A. Key Study

The only study located regarding dermal absorption of HCHs from soil was that of Duff and Kissel (1996) who conducted in vitro dermal absorption studies using human full-thickness skin and two lindane-contaminated soils. The organic content of the sieved sub-150 μm soils were 3.87% (sandy loam) and 0.73% (silt loam). The lindane-spiked soils were stored for up to 19 days prior to testing. No effect of aging was observed within this time frame. The studies were carried out for 24 hours with soil loading at 1, 5 or 10 mg/cm^2 . The relative percent absorption decreased significantly with soil loads of 5 and 10 mg/cm^2 . This was attributed to monolayer coverage of skin occurring at about 2 mg/cm^2 , resulting in reduced fractional absorption at the higher soil loadings.

Results of this study showed that most of the mass of absorbed lindane was found in the skin. The average fraction of total dermal uptake found in the receptor fluid for both soils was only about 4%. Mean 24-hour total dermal absorption values (found in receptor fluid + skin) at a soil load of 1 mg/cm^2 was 1.96 and 2.35%, for low and high organic content soil, respectively. Approximately 40% of the lindane was lost to volatilization with a soil load of 1 mg/cm^2 , while significantly lesser amounts were lost in the higher loading trials (less than 10% for the sandy loam soil at 10 mg/cm^2 ; less than 20% for the silt loam soil at 10 mg/cm^2).

B. Supporting Studies

Feldman and Maibach (1974) examined the percutaneous absorption of lindane dissolved in acetone and applied to the skin of human subjects ($n = 6$). Radiolabeled lindane (4 $\mu\text{g}/\text{cm}^2$) was applied to ventral forearm skin and the urinary excretion of ^{14}C was measured for 5 days after the single topical application. The skin sites were not protected and subjects were asked not to wash the area for 24 hours. Data obtained after i.v. dosing were used to correct the skin penetration data for incomplete urinary recovery. Results indicate that 9.3% (SD 3.7) of the dose was absorbed. However, when skin was occluded, the percent of absorbed dose increased dramatically to 82.1%.

In another human study, lindane was dissolved in acetone and applied to the ventral forearm of volunteers and covered with a nonocclusive patch (Dick et al.,

1997a). Six hours after application approximately 80% of the applied lindane dose (120 mg lindane per ml acetone) had not been absorbed and 14% of the dose was found in the stratum corneum (measured by tape-stripping). The authors conclude that 5% of the applied dose was absorbed to the systemic circulation by 6 hours. Although the disappearance method was used to estimate systemic absorption, measurable levels of lindane were found in the bloodstream and lindane metabolites were found in the urine. By 24 hours, tape stripping of the remaining volunteers showed the stratum corneum contained very little of the applied lindane and only about 0.01% of the dose had been lost through desquamation, suggesting that nearly all the lindane detected in the stratum corneum at 6 hours had been systemically absorbed or absorbed into deeper skin layers by 24 hrs.

F.4.5.2 Discussion and Recommendation for a Hexachlorocyclohexane ABS

Although only one study for dermal absorption of lindane from soil is available, the findings provided consistent results for a human in vitro fractional absorption range of 0.45 to 2.35% under different soil loadings and soil types (Duff and Kissel, 1996). The highest fractional absorption of 2.35% was chosen as the basis for the HCH ABS, given that the soil loading (1 mg/cm^2) used was the only one that was at or below monolayer skin coverage. An average of only 4% of the absorbed dose (approximately 0.09% of the applied dose) was found in the receptor fluid after 24 hrs. However, in vivo studies show extensive absorption of lindane into all skin layers, with continued absorption of lindane beyond the stratum corneum 6 hrs after removal of lindane from the skin surface (Dick et al., 1997a). Thus, lindane retained in skin depots should be presumed to be available for eventual systemic absorption.

Duff and Kissel (1996) noted the unexpected result that the soil with the higher organic carbon content generated a higher fractional absorption (2.35%) than the soil with low organic carbon content (1.96%) at equivalent soil loadings of 1 mg/cm^2 . Increasing organic carbon content of soil generally reduces transport, and dermal absorption, of organic compounds in soil. The authors theorized that this inconsistent finding at 1 mg/cm^2 was due to inter-individual differences in skin absorption, which would not have occurred had the same skin donors been used for both soils.

To account for known effects of organic content of soil the ABS of 2.35% is rounded up, rather than down, to one significant figure for a final ABS of 3%. In support of this ABS adjustment, soil loadings of 5 and 10 mg/cm^2 from high organic content soil did reduce fractional absorption of lindane compared to lindane in soil with low organic content (Duff and Kissel, 1996). However, monolayer coverage of skin was exceeded at these higher soil loads, resulting in lower fractional absorption compared to fractional absorption at 1 mg/cm^2 .

Other data available on percutaneous absorption of lindane or other HCH isomers, which are obtained from studies that use acetone or topical creams and lotions as the vehicle, are not relevant for estimating fractional absorption of lindane from soil (Franz et al., 1996). Use of topical creams and lotions as a vehicle for lindane in dermal absorption studies is related to lindane's use as a medicine to treat scabies.

Theoretical calculations in which release from soil is not the primary limiting factor in the dermal absorption of lindane predict the percent absorbed at 55.6 to 98.5% (Bunge and Parks, 1997). The upper end of this range brackets the 82.1% absorption of applied dose observed by Feldman and Maibach (1974) when the vehicle is acetone and evaporation of lindane is limited by occlusion. However, the lower dermal absorption of lindane from soil observed by Duff and Kissel (1996) is consistent with the theory of slow soil release kinetics, in which partitioning from soil to skin is the limiting factor in dermal absorption for a number of organic compounds (Bunge and Parks, 1997). Oral bioavailability data for absorption of lindane from soil supports the dermal data for absorption of lindane from soil. Soil (organic matter content of 9.8%) spiked with lindane and aged was found to have an oral bioavailability of only 7.2% in an in vitro gastrointestinal extraction test (Scott and Dean, 2005).

The dermal exposure scenario used in this document assumes that deposition of contaminated soil occurs on non-occluded skin exposed to the environment. These conditions would promote evaporation of lindane from soil on the skin, resulting in less absorption into skin than might be expected (Wester and Maibach, 1985; Duff and Kissel, 1996). A potential limitation of this ABS is if significant dermal deposition of lindane-contaminated soil occurs on skin under clothing. The situation may then become one of a reservoir for lindane in which enhanced dermal absorption occurs because of limited evaporation. However, the volatilization potential for lindane from soil also suggests that the absorption potential for lindane may be more significant when exposure is from excavated soils or from surface soils soon after the contamination event (Bunge and Parks, 1997). These various countervailing influences on dermal absorption of lindane under the exposure scenario support the assumption that the ABS will not underestimate actual dermal absorption.

F. 4.6 Diethylhexylphthalate (DEHP)

Recommend point estimate for dermal uptake from soil: 9%

F. 4.6.1 Studies Considered

A. Key Studies

No studies were located on dermal absorption of di(2-ethylhexyl)phthalate (DEHP) from soil.

Deisinger et al. (1998) estimated the migration and subsequent absorption of radiolabeled DEHP from polyvinyl chloride film into rat skin in vivo. Based on the amount of DEHP that migrated from film (505.6 mg) with 24 hr dermal exposure, systemic absorption was estimated at 3.4% of the migrated dose. After skin washing, the residual fraction in skin at the site of dermal application was 13.8% of the migrated dose. Assuming the fraction of DEHP in skin will be eventually absorbed systemically, a maximum absorption rate of $0.24 \mu\text{g}/\text{cm}^2/\text{hr}$ was calculated.

Barber et al. (1992) carried out an in vitro DEHP dermal exposure study to compare rates of absorption through full thickness rat skin and human stratum corneum. DEHP was applied to skin samples in saline solution, and absorption expressed in terms of absorption rate after 32 hrs of exposure. Absorption through rat skin and human stratum corneum was 0.42 and $0.10 \mu\text{g}/\text{cm}^2/\text{hr}$, respectively, indicating that DEHP more rapidly penetrated rat skin than human stratum corneum by a factor of 4.2.

Damage to the rat skin observed following exposure was implied as a possible reason for greater permeability of DEHP through rat skin. Scott et al. (1987) also compared absorption rates of DEHP through rat and human epidermal membranes (dermal layer removed), obtaining rates of 2.24 and $1.06 \mu\text{g}/\text{cm}^2/\text{hr}$ for rat and human skin, respectively. DEHP was applied to the skin sample in 50% v/v aqueous ethanol with exposure up to 53 hrs for rat skin and 72 hrs for human skin. Damage to rat skin, but not human skin, was also observed by Scott et al. (1987) after exposure.

B. Supporting Studies

The National Toxicology Program investigated the dermal absorption of ^{14}C -labeled DEHP in male F344 rats (Melnick et al., 1987; Elsisi et al., 1989). The labeled compound was dissolved in ethanol and applied directly to the skin ($30 \text{ mg DEHP}/\text{kg}$ body weight; $n = 3$ per time point) at a dose of $5\text{-}8 \text{ mg}/\text{cm}^2$. The ethanol was then evaporated and the site of application was covered with a perforated plastic cap. DEHP showed a very slow rate of excretion over five days, likely reflecting a slow dermal uptake process. After five days, approximately 86% of the applied dose was recovered from the skin at the site of application. However, it was not determined how much of the applied dose remained on the surface of the skin and how much was absorbed into the skin. Approximately 5% of the applied dose was recovered in urine and feces, while the amount of the label remaining in the body five days after dosing was less than 2% of the applied dose of DEHP.

Ng et al. (1992) examined dermal absorption of DEHP both in vivo and vitro in hairless guinea pigs. In an in vivo study, radiolabeled DEHP dissolved in acetone ($53 \mu\text{g DEHP}$; $34 \text{ nmols}/\text{cm}^2$) was applied topically on a dorsal area of

the animals which was then covered with a nonocclusive patch. After 24 hours, the patch was removed and the dosing site cleaned to remove any unabsorbed compound. Absorption (estimated from urine and feces) was monitored up to 7 days post treatment. To account for incomplete excretion after the compound was absorbed, a dose of ^{14}C -DEHP was given intramuscularly to a group of animals ($n=5$) and radioactivity was measured in urine and feces for up to seven days.

After 24 hours, 3% (7% after correction) of the dermally applied dose was eliminated in urine and feces. After seven days, approximately 21% (53% after correction) of the dose had been absorbed by the skin and eliminated, while another 11.3% of the dose had been skin stripped from the dose area. An additional group ($n=6$) of animals was given DEHP (53 μg) dermally to estimate the dose remaining in the tissues. After 7 days, ^{14}C content (% of applied dose) was as follows: urine, 18 ± 4 ; feces, 4 ± 1 ; skin wash after 24 hrs, 32 ± 10 ; skin patch, 13 ± 5 ; skin (dosed area), 5 ± 3 ; other tissues (liver, fat, muscle, skin), $4 \pm 3\%$. An additional 10% was estimated to be lost to volatilization.

In the in vitro study, Ng et al. (1992) examined absorption of DEHP through viable and non-viable dermatomed guinea pig skin (200 μm sections) with 24-hr exposure. Radiolabeled DEHP was applied in 10 μl acetone at concentrations of 35.6, 153, or 313 nmol/cm^2 . The percentage of dose that permeated the viable skin into the receptor fluid was 6, 2.4, and 2.5% for the low-, medium-, and high-dose groups, respectively. The percentage of dose that remained in the skin disc was 41.0, 37.5, and 36.2% for the low-, medium-, and high-dose groups, respectively. Use of nonviable skin resulted in a slightly decreased penetration of 5.0% at the applied dose of 35.6 nmol/cm^2 , likely due to decreased metabolism of DEHP. There was a dose-related increase in metabolism but the total metabolites were between 0.5 and 1% of the applied dose for each dose group.

Chu et al. (1996) examined the skin reservoir effects of ^{14}C -labelled DEHP (119-529 $\mu\text{g}/\text{cm}^2$) applied on hairless guinea pigs for 24 hrs, followed by washing of the skin to remove DEHP and analysis of DEHP distribution up to 14 days post-treatment. As DEHP in the dosed skin decreased from 11.1% to 0.66% from 24-hrs to 7 days post-treatment, excreted DEHP gradually increased from 0.74 to 17.3%.

This finding provided evidence that DEHP stored in skin enters the systemic circulation, although the considerable intraspecies variation for percent of absorbed dose precluded a specific estimate of DEHP absorbed systemically after 24 hrs post-treatment. DEHP in the carcass was 1.01 and 0.92% of applied dose at 24 hrs and 7 days, respectively. By 14 days post-treatment, essentially no DEHP remained in dosed skin. Autoradiographic analysis of the dosed skin at 24 hrs revealed dense radiolabel accumulation in the epidermis and along the

hair follicles, which indicated hair follicles may be a penetration pathway for DEHP.

The authors also reported that the percent absorbed at 24 hours by Ng et al. (1992) was higher than that found in this study, with nearly identical experimental protocols. They attributed this difference to the higher doses used in the present study (10 times higher when expressed in $\mu\text{g}/\text{cm}^2$) stating that saturation might have occurred at higher doses, resulting in a lower fractional absorption.

F. 4.6.2 Discussion and Recommendation for a Diethylhexylphthalate ABS

Although two in vitro dermal absorption studies have been carried out with pure DEHP on human skin, data were not provided to determine ABS values. However, absorption rates were determined for both rat and human skin under similar exposure conditions and compared. The DEHP absorption rate for humans was 2-4 times less than that for rats (Scott et al., 1987; Barber et al., 1992).

In vivo studies in rats and guinea pigs that determined absorption of DEHP by total mass balance provide the best estimates for fractional dermal absorption in these species. Deisinger et al. (1998) used PVC film as the vehicle for transfer of DEHP to the skin of rats. Using PVC film as the vehicle will slow absorption, as DEHP requires transfer from the film before partitioning into skin can occur. This type of chemical transfer may give a closer estimate of a DEHP ABS from soil, compared to skin application of the pure compound as performed by the other studies. Including both systemic absorption and compound in skin at the site of application, a fractional dermal absorption value of 17.2% is attained from the Deisinger study. The rat-to-human absorption rate ratio of 2.1 determined by Scott et al. (1987) is then applied to give a final ABS of 9% (rounded up from 8.6%).

DEHP in the skin is included in this estimate, as Ng et al. (1992) and Chu et al. (1996) found there is significant systemic absorption of DEHP in skin up to 7 or more days after removal of DEHP from the skin surface. For this reason, the rat study by Melnick et al. (1987) was not considered in this assessment. The Melnick study did not wash DEHP off the site of skin application prior to analysis, so it is unknown how much DEHP was on or retained in the skin at the end of the 5 day exposure.

Similar to rats, Chu et al. (1996) also noted that guinea pig skin is considered generally more permeable to chemicals than human skin. Thus it is not unexpected that the rat ABS of 17.2% is within the range of 9.5 to 18.9% (DEHP systemically absorbed + DEHP in skin) determined by the authors in guinea pigs. A limitation for this ABS is that both Ng et al. (1992) and Chu et al. (1996) reported that the percent absorbed in guinea pigs appeared to be higher at low application concentrations, although nearly identical experimental protocols were

used. They attributed this difference to possible skin saturation occurring at higher doses (about 119-529 $\mu\text{g}/\text{cm}^2$), resulting in a lower fractional absorption. If saturation of DEHP in rat skin has occurred in the Deisinger et al. (1998) study, this may result in an underestimation of the fractional absorption value at soil concentrations associated with airborne releases.

No data for dermal absorption of the compound bound to soil was located in the literature. In addition, no oral bioavailability studies for DEHP bound to soil could be found. Thus, no further adjustment of the ABS for absorption from a soil was applied.

F. 4.7 Dermal Absorption Fraction for 4,4' -Methylenedianiline

Recommended use of default organic compound ABS estimate of 10%.

F.4.7.1 Studies Considered

Brunmark et al. (1995) utilized a patch-test method to evaluate dermal exposure and pharmacokinetics of 4,4'-methylene dianiline (MDA) dissolved in isopropanol. Measurements of MDA were made in plasma and urine of the five human volunteers. The extent of absorption was evaluated by measuring the amount remaining in the patch after 1 hour. Determination of MDA remaining in the patch showed 25 to 29% was absorbed. The authors also describe elimination half-lives from plasma and urine.

Workers were monitored for two consecutive weeks in a fiber glass pipe factory for dermal exposure to MDA (diluted with triethyleneamine) using both cotton glove and hand wash monitoring (Brouwer et al., 1998). Urinary excretion of methylene dianiline was also evaluated. Urinary MDA levels correlated well with exposure measurements. Geometric means of daily exposure ranged from 81 to 1783 μg MDA, while 24 hour urine samples ranged from 8 to 249 μg MDA. Given that the Brunmark study identified a urinary half-life of MDA of 7 hours and that the measurements on the hands and forearms of the workers correlated strongly (0.94) with the urinary excretion of MDA, one can roughly estimate that between 10 and 14% of the MDA on the hands and forearms was absorbed by the workers.

MDA was applied in vitro to unoccluded human and rat skin for 72 hrs at a loading of 17.7-40.6 $\mu\text{g}/\text{cm}^2$ in ethanol (Hotchkiss et al., 1993). Absorption into the receptor fluid at 72 hrs was 6.1 and 13.0% of the applied dose for rat and human skin, respectively. When the skin was occluded, the absorption at 72 hrs was significantly enhanced, reaching 13.3 and 32.9% for rat and human skin, respectively. MDA that remained in human skin at 72 hrs was 23.8 and 37.4% of the applied dose for unoccluded and occluded skin, respectively. For the rat, MDA content of the skin at 72 hrs was 57.6 and 53.1% of the applied dose for unoccluded and occluded skin, respectively. Although the data were only

graphically presented, absorption through human skin into the receptor fluid at 24 hrs can be estimated at 8% of the applied dose for unoccluded skin and 20% of the applied dose for occluded skin.

The permeability of rat and human skin in vitro to MDA was assessed by Kenyon et al. (2004) over a large dose range, and the potential for skin to act as a reservoir for MDA was investigated. Dose levels of 0.01, 0.1 and 1 mg per 0.32 cm² skin were applied in ethanol:water (50:50) onto occluded skin for 24 hrs. No statistical difference in skin permeability was observed between rat and human skin. After 24 hrs, 27 to 52% of applied MDA had penetrated human skin to the receptor fluid. The percentage of applied MDA retained in human skin was 20%.

In another in vitro experiment, Kenyon et al. (2004) applied 0.1 mg MDA to human skin for 4 hrs, then removed excess MDA on the skin surface and the experiment continued for another 4 hrs. The cumulative absorption rate of MDA into the receptor fluid remained the same for the last 4 hrs, with only a slight decrease noted between 7 and 8 hrs. Of the total 11% of the MDA found in the skin, 5% was removed by tape stripping the stratum corneum. The remaining 6% of MDA were found in the digested skin, suggesting this amount would have been absorbed had the experiment continued longer. Considering that the lag time for appearance of MDA in receptor fluid was about 4 hrs, the authors presumed that the MDA remaining in the stratum corneum at 8 hrs would not be absorbed systemically.

No literature could be located regarding dermal absorption of MDA from soil. However, the fate of MDA added to soil has been investigated. MDA rapidly and strongly absorbs to loam soil which contained a total organic content of 1.3% (Cowen et al., 1998). However, MDA does not appear to form complexes with humic materials or form other irreversible soil binding processes. In one year, the aerobic biodegradation of MDA in silt loam soil was 40%.

F.4.7.2 Discussion and Recommendation for a 4,4' -Methylenedianiline ABS

Dermal absorption of MDA in workers is considered a more significant route of exposure than inhalation (Brouwer et al., 1998). The in vivo worker data support the in vitro human data in that dermal absorption is considerable. However, the exposure/application of MDA involved other organic solvents. The effect of solvent vehicle on absorption was not investigated.

No data could be located regarding dermal or oral absorption of MDA bound to soil. In addition, no oral bioavailability studies for MDA bound to soil could be located. Soil fate studies indicate that MDA binds strongly to soil, which would likely reduce dermal absorption considerably, and biodegrades slowly over a year's time. Thus, the default absorption value of 10% for organic compounds is recommended until soil-bound dermal studies are available.

F.5 Comparison with Other Published Dermal Absorption Factors

Two other agencies have published fractional dermal absorption estimates for some of the Hot Spots chemicals presented in this document. These values are shown in Table F.5 and are compared with the fractional dermal absorption values developed by OEHHA.

Table F.5. Published Point Estimates and Default Dermal Absorption Factors (ABS) as Percent of Selected Chemicals from Soil

CHEMICAL	ABS (percent)		
	OEHHA ^a	US EPA ^b	DTSC ^c
Inorganic chemicals			
Arsenic	6	3	3
Beryllium	1	^d	^e
Cadmium	0.2	0.1	0.1
Chromium (VI)	2	^d	^f
Fluoride	1	^d	^e
Lead	3	^d	^e
Mercury	4	^d	^e
Nickel	2	^d	^e
Selenium	1	^d	^e
Organic chemicals			
Di(2-ethylhexyl)phthalate (DEHP)	9	^h	^h
Hexachlorobenzene	4	^h	^h
Hexachlorocyclohexanes (as lindane)	3	^h	^h
4,4'methylene dianiline (MDA)	10	^h	^h
Polychlorinated biphenyls (PCBs)	14	14	15
Polychlorinated dibenzo-p-dioxins and dibenzofurans (as TCDD)	3	3, 0.1 ^g	3
Polycyclic aromatic hydrocarbons	13	13	15

^a ABS values, as presented in this document by OEHHA. In most cases, the OEHHA ABS represent dermal absorption values based on the soil vehicle freshly spiked with the chemical contaminant and placed on skin for up to 24 hrs.

^b (U.S. EPA, 2004)

^c (DTSC, 1994)

^d An ABS point estimate is not specifically listed for this chemical. For inorganics with insufficient data, USEPA (2004) states that the speciation of the compound is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value.

^e California's Department of Toxic Substances Control (DTSC, 1994) recommends using 1% as the default dermal absorption value for metals, based on Clement Associates (1988).

^f California's Department of Toxic Substances Control (DTSC, 1994) in their Preliminary Endangerment Assessment Guidance Manual does not recommend a fractional absorption value for Cr(VI) due to lack of systemic carcinogenicity via non-inhalation routes of exposure.

^g USEPA (2004) recommends a dermal absorption fraction from soil of 3%, or a dermal absorption fraction of 0.1% if the soil organic content is > 10%.

^h No specific default ABS value is listed, although a default dermal absorption fraction for semivolatile organic compounds (SVOCs) of 10% as a screening method is used for the majority of SVOCs without dermal absorption fractions.

F.6. References

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Appendix G
Chemical Specific Soil Half-Lives

Appendix G

Chemical-specific Soil Half-life

The average concentration of a substance in soil (C_{soil}) is a function of several different variables, including deposition rate, accumulation period, mixing depth, soil bulk density, and the chemical-specific half-life, as shown in equation G-1 below:

$$C_{soil} = [GLC (Dep\text{-}rate) (86,400) (X)] / [K_s (SD) (BD) (T_t)] \quad (\text{Eq. G-1})$$

where: C_{soil} = average soil concentration at a specific receptor location over the evaluation period ($\mu\text{g/kg}$)
GLC = ground level concentration from the air dispersion modeling ($\mu\text{g/m}^3$)
Dep-rate = vertical rate of deposition (m/sec) (see Chapter 2 for values)
86,400 = seconds per day conversion factor
 X = integral function accounting for soil half-life
 K_s = soil elimination time constant = $0.693/T_{1/2}$
SD = soil mixing depth = 1 cm for dermal scenario
BD = bulk density of soil = 1333 kg/m^3
 T_t = total averaging time = 70 years = 25,550 days

The soil half-life is part of the integral function X determined as below:

$$X = \{[\text{Exp} (-K_s \times T_f) - \text{Exp} (-K_s \times T_0)] / K_s\} + T_t \quad (\text{Eq. G-2})$$

where: EXP = Exponent base $e = 2.72$
 K_s = soil elimination constant = $0.693/ T_{1/2}$
 $T_{1/2}$ = chemical-specific soil half-life
 T_f = end of exposure duration (days); 25,500 for a 70-year exposure
 T_0 = beginning of exposure duration (days) = 0 days
 T_t = total days of exposure period = $T_f - T_0$ (days)

Estimating toxicant soil concentration is necessary for estimating dose from incidental soil ingestion by home raised meat, home raised produce, and dermal absorption via contact with contaminated soil.

Since the chemicals that the Hot Spots program is concerned with are emitted into the air and then subject to deposition to the soil, there are only two classes of chemicals considered. These classes are the semivolatile organic chemicals, such as PAHs, PCBs and dioxins, and toxic metals such as hexavalent chromium, cadmium, lead, arsenic, and beryllium. Other programs that consider hazardous waste sites may be concerned with other classes of chemicals such volatile organic solvents.

Soil extraction studies were often used to estimate soil half-life by using rigorous extraction techniques with an organic solvent (e.g., dichloromethane) to release as

much of the remaining chemical from soil as possible. The amount of chemical extracted from soil is considered the fraction that is bioaccessible for uptake. The bioaccessible fraction of a pollutant in soil, which is reduced over time by various processes, is used to estimate the soil half-life of chemicals.

An extraction procedure that mimics or parallels bioavailability is preferable for assessing exposure and risk than one whose sole virtue is the removal of the largest percentage of the compound from soil (Kelsey, 1997; Reid, 2000; Tang, 1999). This investigation suggests that mild, selective extractants may prove more useful as predictors of exposure than the methods currently used for regulatory purposes in some programs. The solvent needed for predictive purposes may vary with the pollutant and the species of concern.

Another common method to determine soil half-life of organic compounds is through mineralization, or ultimate degradation, studies. Instead of measuring the parent organic compound remaining in soil through extraction methods, mineralization studies add the radiolabeled chemical to soil, and measure the release of $^{14}\text{CO}_2$ from soil resulting from “ultimate” breakdown of the compound by microbial degradation.

Mineralization studies may be quite useful for determining the soil half-life of organic chemicals, if abiotic loss processes are minor, and if mineralization of the chemical occurs quickly once primary degradation (and presumably loss of toxicity) of the chemical takes place.

G.1 Metals

Biodegradation as such is not expected to occur with metals and other elements because of their elemental nature. However, once a metal is deposited to soil, leaching or weathering may eventually result in movement of the metal out of the system. The valence and charge of the metal in soil affects their sorption, solubility, and retention in soil. Additionally, soil pH and availability of charged sites on soil surfaces are the primary factors controlling formation of the ionic species, charged metal complexes or precipitates (US EPA, 2003).

Soil with predominately negatively charged sites is more plentiful in the United States; less than 5% of the total available charge on the soil surface is positively charged (US EPA, 2003; Fairbrother et al., 2007). For the metals that largely exist as cations in soil (beryllium, cadmium, lead, inorganic mercury and nickel), there is a greater propensity to be sorbed to soil particles. This makes them less bioavailable, but it also results in greater loading of metals into the soil because of reduced mobility and leaching.

Under most relevant scenarios, arsenic, chromium and selenium deposition to soil, typically results in formations of anionic complexes with oxygen (US EPA, 2003; Fairbrother et al., 2007). The most common forms of arsenic are arsenate (As(V)) and arsenite (As(III)), which are present in soil solution in the form of AsO_4^{3-} and AsO_3^{3-} ,

respectively. Similarly, selenium can be present as selenates (SeO_4^{2-}) and selenites (SeO_3^{2-}). Hexavalent chromium (Cr(VI)) can exist as chromate (CrO_4^{2-}) which is usually considered more soluble, mobile, and bioavailable than the sparingly soluble chromite (Cr(III)), which is normally present in soil as the precipitate $\text{Cr}(\text{OH})_3$. Anionic metals generally move into pore water where they can leach out of the system faster, but are also more bioavailable.

As a default estimate, the metal content of soil is assumed to decay with a half-life of 10^8 days unless site-specific information is presented showing that soil conditions will result in the loss of soil metal content, i.e., soil aging or leaching. The 10^8 default means that significant loss or removal is not occurring within the risk assessment time frame of interest.

Some fraction of chromium (VI) will undergo reduction to the less toxic chromite (Cr(III)) species when deposited to soil (Bartlett, 1991; Fendorf, 2004; Stewart et al., 2003). However, oxidation reactions of chromium (III) to chromium (VI) can also occur at the same time in soil. Characterizing the reduction of chromium (VI) to chromium (III) is complex and "it is not possible to predict how chromium compounds will behave in soil until the soil environment has been adequately characterized" (Cohen et al., 1994a, citing Gochfeld and Whitmer, 1991). Several tests have been suggested for evaluating the reducing capacity of soils and may be considered in the development of site-specific information (Cohen et al., 1994a, citing Bartlett and James, 1988; Walkley and Black, 1934). These tests are described as follows:

"(1) Total Cr(VI) Reducing Capacity. Use the Walkley-Black (1934) soil organic matter determination in which carbon oxidizable by $\text{K}_2\text{Cr}_2\text{O}_7$ is measured by titrating the Cr(VI) not reduced by a soil sample (in suspension with concentrated H_2SO_4) with $\text{Fe}(\text{NH}_4)_2(\text{SO}_4)_2$.

(2) Available Reducing Capacity. Shake 2.5 cm^3 of moist soil 18 hours with 25 mL of 0.1 to 10mM chromium as $\text{K}_2\text{Cr}_2\text{O}_7$ in 10mM H_3PO_4 , filter or centrifuge, and determine Cr(VI) not reduced in the extract by the s-diphenylcarbazide method.

(3) Reducing Intensity. The procedure is the same as that used in (2) above except that 10mM KH_2PO_4 should be used in the matrix solution in place of H_3PO_4 ."

In the absence of site-specific data, the public health protective assumption is to assume that hexavalent chromium remains in the hexavalent form in the soil. In most instances this will lead to an over prediction of hexavalent chromium concentration from airborne deposition.

G.2 Organics

Organic compounds deposited in soil are subject to degradation or loss by both biotic and abiotic processes. Biotic processes include degradation by soil microorganisms. Abiotic loss of organic compounds in soil includes such processes as photochemical reactions (if on the surface of the soil) or volatilization from the soil.

For some persistent organic chemicals, such as PAHs, soil aging is the abiotic process causing the most loss. Aging is associated with a continuous diffusion and retention of compound molecules into remote and inaccessible regions within the soil matrix over time, often on the order of weeks or months, thereby occluding the compounds from abiotic and biotic processes (Northcott and Jones, 2001).

Many earlier soil half-life studies assumed that decreased soil extractability and bioavailability of chemicals with time was due to biodegradation by soil microorganisms, when, in fact, soil aging is a significant or dominant factor. Soil aging represents an abiotic loss process in which chemicals in soil become inaccessible for microbial degradation. Soil half-life of an organic compound can vary to a large extent depending on pre-treatment of soils before or after addition of the chemical to soil, the methodology used for soil extraction of the compound, and soil organic content. Other variables that can influence a soil half-life include vegetation coverage, weather and climate, and the presence of co-contaminants.

The organic carbon content of soil is often a major factor influencing the half-life of an organic compound. Increasing the organic carbon content of soils will increase sequestration and decrease bioavailability of organic chemicals. The amount of organic material in the soil is expressed as either organic carbon or organic matter. A conversion factor of 1.724 can be used to approximate the OC content of a soil that is expressed as OM (Northcott and Jones, 2001). The OC or organic matter (OM) contents of the soils used are identified in the summaries below if included in the study methodology. The OC content of the contaminated soil at a particular site can be taken into consideration if enough data is present to show that the OC content is a significant factor for the soil half-life of an individual chemical. A default assumption is available for the Hot Spots program, in which the fraction organic carbon in soil is 10%.

Considerable differences between field and laboratory half-life estimates have also been found for some organic chemicals such as PAHs (Doick et al., 2005). Pollutant fate studies are frequently performed under laboratory conditions and over short time periods. Field studies under realistic environmental conditions and protracted time frames probably represent a better estimate of the soil half-life and, therefore, carry more weight for estimating the soil half-life.

G.2.1 Creosotes

Creosotes are of concern primarily because of the polycyclic aromatic hydrocarbon content, which represent 85-90% of creosote constituents (Cerniglia, 1992). Therefore, in terms of soil half-life of this complex mixture, OEHA recommends using the PAH half-life of 429 days for creosotes (see below).

G.2.2 Diethylhexylphthalate

Phthalates share the same basic structure of an esterified benzenedicarboxylic acid with two alkyl chains, and are chemically stable in the environment (Cartwright et al., 2000; Staples et al., 1997). Thus, the general absence of high concentrations of phthalates in the environment indicates the importance of biodegradative processes, specifically those mediated by microorganisms because higher organisms are unable to cleave phthalate's aromatic ring.

Metabolism of DEHP often results in the formation of the MEHP and phthalic acid. These metabolites retain some toxicological properties but are metabolized at a much faster rate than DEHP. Therefore, mineralization (i.e., ultimate degradation) of DEHP represents a reasonable and health protective indicator of the destruction of the phthalate's toxicological potential (Maag and Lokke, 1990). The very high Koc and Kow values for DEHP relative to other phthalates promotes slower degradation in soil because a major fraction of this compound can eventually become strongly sorbed to soil organic material (i.e., soil aging) and therefore becomes much less bioavailable to soil microorganisms (Gejlsbjerg et al., 2001; Madsen et al., 1999).

Numerous microbial DEHP degradation studies are available in the literature, many of which measured degradation in unadulterated agricultural/garden soil. Only two studies were located in which DEHP soil degradation was investigated outdoors. In one study, DEHP-polluted sandy soil was mixed with compost topsoil and fertilizer, and then layered over a grass-covered plot (Maag and Lokke, 1990). White clover and grass were sown into the plot with four soil samples collected for analysis over 192 days. The depletion of extracted parent compound from soil roughly followed first-order kinetics with a half-life of 73 days.

In the other outdoor study, [¹⁴C]DEHP was applied to sandy soil (pH 6.8, organic matter 0.3%) and potatoes planted the first year, followed by planting of barley during the second year (Schmitzer et al., 1988). Only 6.9% of the applied radiocarbon, mainly as DEHP, was recovered after 111 days when the potatoes were harvested. Nearly all the remaining activity, at least 92.3%, was lost to the atmosphere as ¹⁴CO₂. After 446 days when the barley was harvested, only 1.7% of the radiocarbon was found in the soil. A half-life was not determined, although assuming first order kinetics, the half-life would roughly be 30 days over the first four months of the study.

In a highly detailed laboratory study, Madsen et al. (1999) revealed that there are actually two phases in the mineralization of [^{14}C]DEHP in a sandy loam soil (pH 5.9, OC 2.5%) over a 130 day exposure - an initial phase during the first 30-60 days described well by first-order kinetics, and a late phase in which mineralization activity was much lower. This second phase was thought to represent mineralization that was increasingly regulated by strong sorption to organic matter, resulting in decreased bioavailability to soil microbes. The researchers also observed mineralization was strongly regulated by temperature, with the rate of mineralization increasing with increasing temperature. To account for diurnal swings in temperature that would occur in the field, the mean half-life over the temperature range examined (5, 10 and 20 °C) was 99 days during the initial phase and 161 days during the late phase.

A similar two-phase degradation rate for [^{14}C]DEHP was observed by Roslev et al. (1998) in a sludge-amended soil (DEHP is a common contaminant in sludge). The half-life for mineralization in a sandy loam soil (pH 5.9, organic matter 2.5%) was found to increase 2.5-fold in the late phase from 58 to 147 days.

Slow degradation of DEHP has been observed in other laboratory studies. Cartwright et al. (2000) observed that only 10% of DEHP added to a sandy clay loam soil (pH 6.25, OC 3.78%) was removed by indigenous microbes by day 70. Gejlsbjerg et al. (2001) observed an average mineralization of [^{14}C]DEHP in three Danish agricultural soils (pH 6.0-6.6, OC 2.2-3.0) to be only 13% (range = 8.46 to 21.8%) over two months. In both studies, strong sorption to soil organic matter was assumed to be the reason for slow microbial degradation.

On the other hand, rapid soil degradation of DEHP has also been observed. Kirchmann et al. (1991) determined a half-life of 20-80 days for loss of parent DEHP extracted from soil (pH 7.3, OC 1.77%), although the data suggested more of a linear disappearance of DEHP with time, rather than a first order disappearance. Shanker et al. (1985) observed a half-life of 15 days for loss of parent DEHP extracted from garden soil (pH 8.2) under a relatively high incubation temperature (30 °C).

The soil half-life of DEHP can vary greatly depending on the soil conditions, with a significant amount of the parent compound eventually being sorbed to soil organic matter for long periods and becoming recalcitrant to breakdown by soil microbes. The soil half-life of 73 days based on the field study by Maag and Lokke (1990) is used here as the default soil half-life for DEHP. Similar results were obtained in comprehensive soil mineralization studies by Madsen et al. (1999) and Roslev et al. (1998), although first order kinetics were not strictly followed over the full length of the studies.

G.2.3 Hexachlorobenzene

Hexachlorobenzene is a persistent soil contaminant that does not appear to be significantly degraded in soils by either abiotic or biodegradation processes (Isensee et al., 1976; Beall, 1976). In a simulated field experiment conducted in a greenhouse,

HCB applied to soil almost completely volatilized from the first two cm of soil after 19 months. However, only about 20% of the HCB was lost at a soil depth of 2-4 cm over 19 months. Only the parent compound was found in soil throughout the experiment suggesting HCB could be quite stable and persistent in a plowed field. It should be noted that this study used a single addition of HCB to the soil and the distribution of HCB with long-term low level (deposition) is likely to be different.

A soil half-life estimate for HCB was obtained from a controlled laboratory experiment conducted in plastic-covered pots over a period of 600 days (Beck and Hansen, 1974; Bro-Rasmussen et al., 1970). Analysis for parent compound following soil extraction showed a soil half-life for disappearance of HCB to be 969-2089 days with a mean of 4.2 years. In a similar experiment, Isensee et al. (1976) observed no loss of HCB from soil in covered beakers over a one-year period.

The data show loss of HCB from soil to be primarily by volatilization with essentially no loss due to microbial degradation. It is recommended that as a default estimate, the deposition of HCB to soil in particle form be assumed to decay with a half-life of 10^8 days, similar to the metals.

HCB accumulation in the soil from airborne sources has been shown to occur in field studies. There are a couple of mechanisms that could account for this observation. HCB could partition and bind tightly onto airborne particulate matter and then be subject to deposition. Alternatively, tight binding of gaseous HCB to soil could effectively make the soil a sink for gaseous airborne hexachlorobenzene. The studies in which hexachlorobenzene is added directly to soil establish that hexachlorobenzene below a certain depth remains in the soil, presumably bound.

G.2.3 Hexachlorocyclohexanes

The α - and γ -forms of the HCHs are the most common isomers in technical grade HCH, while the β -isomer is generally the most environmentally persistent. Similar to HCB, loss of HCH deposited on soil is expected to be primarily from volatilization, although some microbial degradation has been shown to occur with the HCHs (Spencer et al., 1988; Jury et al., 1987). HCH tilled into soil will adsorb to soil organic matter significantly reducing the potential for volatilization. HCHs can undergo dehydrochlorination by soil microbes in moist, acidic-to-neutral soils (Yule et al., 1967). Anaerobic soil conditions tend to favor faster degradation over aerobic conditions (MacRae et al., 1984).

No recent soil half-life studies for HCHs conducted in the U.S. could be located. Early field studies in the U.S. suggested a soil half-life for Lindane (γ -HCH) to be on the order of months to years (Lichtenstein and Schultz, 1959; Lichtenstein and Polivka, 1959). However, the method of detection used also included detection of relatively non-toxic degradation products of Lindane. It was also unclear if offsite atmospheric deposition of

HCHs onto the field plots was occurring, which can dramatically increase the apparent half-life of HCHs if not taken into account (Meijer et al., 2001).

Table G.1 Soil half-lives (days) for HCHs in subtropical environments of India.

	Singh et al., 1991 ^a	Kaushik, 1989 ^a	Srivastava & Yadav, 1977
α -HCH	55	-	-
γ -HCH	85	-	-
β -HCH	142	-	-
Technical HCH	-	23	44

^a Half-lives are an average of cropped and uncropped soils

In an Indian field study, Kaushik (1989) monitored the loss of technical grade HCH sown into the top 15 cm of a field that remained fallow, and a field that contained plants and was watered regularly. The climate was characterized as subtropical, and the soil in both fields was sandy loam with a pH of 8.2 and an OC content of 0.8-1.0%. In the fallow field, the HCH half-life in the upper and lower 7.5-cm soil layers was 21 and 41 days, respectively, with a combined total half-life of 26 days. In the planted field, a total half-life of 20 days was recorded, with little difference in HCH loss observed between the upper and lower soil layers field.

In another Indian field study, Singh et al. (1991) determined the soil half-lives for several HCH isomers sown into the top 10 cm of cropped and uncropped sandy loam soil (pH 7.8; OC 0.63%) over a 1051 day period. Half-life values in the subtropical climate showed similar persistence in cropped and uncropped treatments. The longest half-life was observed for β -HCH (100 days cropped; 183 days uncropped) and the shortest half-life was observed for α -HCH (56.1 days cropped; 54.4 days uncropped). Another field study in India observed an average soil half-life of 44 days (range: 35 to 54 days) for a low concentration of technical grade HCH applied under cover of maize crop over three years of planting (Srivastava and Yadav, 1977).

Researchers have noted that the soil half-life for HCHs estimated in tropical climates likely underestimates the half-life for HCHs in cooler, temperate climates of the U.S. due to greater volatility, and probably higher microbial degradation, at warmer temperatures (Singh et al., 1990; Kaushik, 1989). Because temperate climate of California will tend toward lower volatility of HCHs from soil, the longer HCH half-lives determined by Singh et al. (1991) in Table G.1 are recommended for use in the "Hot Spots" program. If the HCH isomer profile in the soil is unknown, an average of the three isomer soil half-lives (94 days) can be used.

G.2.4 4,4'-Methylenedianiline

Cowen et al. (1998) investigated biodegradation of 4,4'-methylenedianiline under aerobic and anaerobic conditions using ¹⁴C labeled methylenedianiline. The data

showed that, after 365 days of aerobic biodegradation in silt loam soil, 59.9% of 4,4'-methylenedianiline remained intact. Based on the aerobic biodegradation data from this study, using first-order kinetics default for dissipation of chemicals, OEHHA derived a soil half life of 455 days for 4,4'-methylenedianiline.

G.2.5 Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a mixture of chlorinated biphenyl congeners that vary in the degree of chlorination. The degree of chlorination has a major impact on soil half life. Several different mixtures were marketed and used widely before PCBs were banned because of their toxicity, environmental persistence and bioaccumulative properties. Small amounts are generated as combustion byproducts and these emissions are subject to the Hot Spots program. The toxicity of individual congeners varies widely. For these reasons, meaningful overall soil half-life for PCBs is difficult to ascertain for situations in which PCB emissions are not speciated and the cancer potency factor for the entire mixture is applied. A half life of 940 days for Aroclor 1254 was derived by Hsieh et al. (1994). This value is used by the Department of Toxic Substances Control in CalTOX. In 2000, OEHHA proposed to use this value for all Aroclor mixtures and airborne emissions of unspciated PCB mixtures generated from Hot Spots facilities.

Harner et al. (1995) studied four PCB congeners (28, 52, 138, 153) in air, herbage, and soil of the southern U.K. over the period 1942-1992 and observed soil half-lives ranging from 7 to 25 years (mean 18 years) (6570 days). Wania and Daly (2002) estimated soil half-lives of seven PCB congeners (8, 28, 52, 101, 153, 180, 194) ranging from 550 hours (23 days) to 1,700,000 hours (70,833 days).

Sinkkonen and Paasivirta (2000) suggested soil half-lives for eleven PCB congeners, ranging from 26,000 hours (1,083 days) to 330,000 hours (13,750 days), based on the work of Lake et al. (1992), Beurskens et al. (1993) and Brown et al. (1984).

Doick et al. (2005) studied long-term fate of two PCBs in an agricultural soil in Germany. Their observation over 152 months concluded that the soil half-lives were 10.9 years (3979 days) for PCB 28 and 11.2 years (4088 days) for PCB 52. The authors attributed the much longer soil half-lives of PCBs than estimates in other studies to length of study, field study conditions, vegetation (type and coverage), weather and climate, the presence of co contaminants and, particularly, soil type -- a high silt, high clay content, "heavy" soil with reduced water infiltration, compared with higher porosity, sandy soils.

There is great variability in soil half-lives among the PCB congeners in the above studies. The OEHHA adopted Toxicity Equivalency Factors (TEF) for individual PCB congeners (WHO97-TEF) (OEHHA 2003a); thus, it is appropriate to apply the soil half life data for these individual congeners where speciation of PCBs has been performed on facility emissions. Based on the studies above, only the data for PCB congeners with a WHO TEF (IUPAC # 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189) were

used for estimating soil half-lives in this document, unless only total PCBs are available (OEHHA 2003b).

Among the above studies, Lake et al. (1992) derived a half-life of 7.5 years for PCB 105 and 6.8 yrs for PCB 118, using the anaerobic dechlorination reaction in sediment of 15-17.5 cm deep from New Bedford Harbor, Connecticut. Beurskens et al. (1993) have estimated a half-life time of nine years for PCB 105, PCB 126, PCB 156 and PCB 169 in the anaerobic sediment. Brown et al. (1984) found the average elimination half-life for PCB 105 and PCB 118 in Hudson river sediments was 10 years. The OEHHA acknowledges that the degree of biodegradation in sediment would be different from that for a dry land scenario. Until studies in dry soil become available, the river sediment data appears to be the best choice.

Table G-2. Soil half-lives (days) for PCBs (IUPAC #) relevant to the “Hot Spots” program

Study	105	118	126	156	169	Total PCBs
Lake et al. 1992	2738	2482				
Beurskens et al., 1993	3285		3285	3285	3285	
Brown et al. 1984	3650	3650				
Arithmetic mean half-lives	3224	3066	3285	3285	3285	

The arithmetic mean half-lives for each PCB are shown at the bottom of Table G-2, and a grand mean half-life including all studied PCBs is 3229 days. This overall half-life of 3229 days is recommended as the estimated soil half-life for PCBs.

G.2.6 Polycyclic Aromatic Hydrocarbons (PAHs)

There are a variety of polycyclic aromatic hydrocarbons emitted from combustion sources. The structures vary by number and placement of fused aromatic carbon rings and functional groups on those rings. In general, it has been observed that the soil half-life increases with the increasing number of fused rings on a PAH and is correlated directly with molecular weight and K_{ow} (Northcott and Jones, 2001; Wild and Jones, 1993). The PAHs currently of toxicological concern under the “Hot Spots” program consist almost entirely of four or more rings with the prototype PAH, benzo(a)pyrene, containing five fused benzene rings. Naphthalene is carcinogenic and only has two rings but it is too volatile to be a multipathway chemical subject to deposition. Therefore, OEHHA chose to base the soil PAH half-life on those compounds with greater than three rings to avoid underestimating the accumulation of the carcinogenic PAHs in the soil.

Studies where PAHs have been added to soil have noted that those PAHs with three rings or less show significant volatilization from soil and microbial degradation, whereas

PAHs with greater than three rings show little or no volatilization and slower microbial degradation (Wild and Jones, 1993; Cerniglia, 1992). In addition, a broad inverse relationship has been observed between the rate of biodegradation and the organic carbon (OC) content of the soil (Northcott and Jones, 2001; Wild and Jones, 1993). Soil half-life estimates for PAHs that currently have a potency equivalency factor (PEF) were given the greatest weight in determining a default soil half-life. Table G-3 shows the PAH half-life results from the most comprehensive studies found in the literature and a brief summary of the studies is given below.

Doick et al. (2005) conducted a field study and determined the long-term fate of ^{12}C and ^{14}C analogues of benzo[a]pyrene spiked in a cultivated agricultural soil subject to typical agricultural practices. The soil had a pH=7.2 and an organic matter content of 2.2%. Their observation over 152 months found that the soil half-life for benzo-[a]pyrene was 2.7 years (982 days). These half-life values are much longer than estimates in other studies and are thought to be a result of the soil type, length of the study, use of field conditions rather than laboratory conditions, and vegetation (type and coverage).

Sewage sludge containing PAHs was applied to two agricultural soils at five dose levels (30 to 600 t/ha) in field plots, followed by cultivation with annual crops or a perennial (willow) for up to 54 months (Oleszczuk and Baran, 2005). It was unclear from the description of the methodology if this work was an actual field study. Before addition of the sewage sludge, the soil with the annual crops had a pH=4.3 and a total organic carbon (OC) content of 1.12%. The soil with the perennials had a pH=5.8 and a total OC content of 1.21%. Analysis of 16 PAHs showed longer half-lives in the soil with the annual crops. However, the sewage sludge properties were considered as important as the type of crop used. The investigators suggested that longer half-lives of PAHs compared to other studies may have occurred due to the increased soil aging process in a soil-sludge matrix.

In a climate-controlled greenhouse experiment, sewage sludge containing PAHs was applied to four different soils to determine the soil half-life for a number of individual PAHs (Wild and Jones, 1993). The four soils ranged from a sandy clay loam agricultural soil (pH=6.6, organic carbon content, 6.04%) to a coniferous forest soil (pH=2.9, organic carbon content, 58%). Although the half-lives among 12 PAHs measured in the forest soil tended to be longest, the overall average of the sum of the PAH half-lives was not considerably higher in forest soil ($t_{1/2}$ =192 d) compared to the overall average of the sum of the half-lives in the agricultural soils ($t_{1/2}$ =146 d and 165 d) and a roadside soil (177 d). The authors noted that the controlled environmental conditions in the greenhouse optimize biodegradation compared to field conditions, and likely results in more rapid losses of PAHs from the soil.

Two different sandy loam soils were spiked with 14 PAHs in incubation chambers and their soil half-lives estimated over an exposure period of up to 196 days (Park et al., 1990). One soil (Kidman sandy loam) had a pH=7.9 and an OC content of 0.5%, and the other soil (McLaurin sandy loam) had a pH=4.8 and an OC content of 1.1%. The

half-lives for PAHs with PEF values ranged from 24 days to 391 days. Although the organic content and pH of the two soils differed, the biological degradation rates of the PAH compounds were not statistically different between the two soils.

In another laboratory study, Coover and Sims (1987) spiked a sandy loam agricultural soil (pH=7.9; OC content, 0.5%) with 16 PAHs and estimated the soil half-lives over a 240 day incubation period. Increasing the soil temperature was observed to increase the apparent loss of low molecular weight PAHs but had little effect on loss of five- and six-ring PAHs.

Table G-3 Soil half-lives (days) for PAHs relevant to the “Hot Spots” program

Study	Ch	BaA	BaP	BbF	BkF	DahA	DaiP	Ind	DaA
Coover & Sims, 1987 ^a	1000	430	290	610	1400	750		730	
Park et al., 1990 ^b	379	212	269	253		391	297	289	24
Wild & Jones, 1993 ^c	215	215	211	202	301				
Doick et al., 2005			982						
Arithmetic mean half-lives	531	286	438	355	851	571	297	510	24

Abbreviations: Ch, chrysene; BaA, benz[a]anthracene; BaP, benzo[a]pyrene; BbF, benzo[b]fluoranthene; BkF, benzo[k]fluoranthene; DahA, dibenz[a,h]anthracene; DaiP, dibenzo[a,i]pyrene; Ind, Indeno[1,2,3-c,d]pyrene; DaA, 7,12-Dimethylbenz[a]anthracene

^a Environmental temperature held at 20C

^b Average half-life values for two sandy loam soils

^c Average half-life values for four different soils. Ch and BaA co-eluted; the $t_{1/2}$ is for both PAHs combined

The arithmetic mean half-lives for each PAH are shown at the bottom of Table G.3, and a grand mean half-life including all PAHs is 429 days. Greater differences in PAH half-lives are seen between studies rather than within studies. One possible reason is that longer half-lives are attained from field studies (Doick et al., 2005) compared to laboratory studies (Coover & Sims, 1987; Park et al., 1990; Wild & Jones, 1993).

However, the limited number of field studies makes it difficult to confirm this assumption. The overall PAH half-life of 429 days is recommended until further field studies are conducted.

G.2.7 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans (PCDD/F)

The prototype compound and most potent of the dioxin and furan family of compounds is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The degree and placement of chlorination affects both the toxicity and soil half life of dioxins and furans. Sampling of 32 sites in Seveso, Italy, produced an initial calculated regression half-life of one year (365 days) (Di Domenico et al., 1980). Experimental application of TCDD to two different soil types (loamy sand and silty clay loam) for 350 days produced calculated half-life values ranging from 394 to 708 days (Kearney et al., 1972; Kearney et al., 1973). Soil half-life estimates ranging from 10 to 12 years (3650-4380 days) were reported based upon experimental measured soil concentrations of TCDD from a contaminated site at an Air Force base in Florida (Young, 1981). Soil half-life estimates of 10 to 100 years (3650-36500 days) were reported, depending on the depth of the contamination, with deeper soil having reduced biodegradation rates (Nauman and Schaum, 1987). An estimated soil half-life of 3609 days has also been reported (calculated from a soil reaction rate constant of $8 \times 10^{-6} \text{ hr}^{-1}$) (Mackay et al., 1985).

Several other half-life estimates have also been identified and summarized (Cohen et al., 1994b). Soil samples showing loss of TCDD content by volatilization produced estimated half-lives of 7-24 days (Nash and Beall, 1980). TCDD measured in soils from the contaminated site in Seveso, Italy, produced a half-life estimate of 9.1 years (3322 days) (Cerlesi et al., 1989). A half-life estimate of 3 days was made based on loss of TCDD content from soil by both photodecomposition and volatilization (Di Domenico et al., 1982).

McLachlan et al. (1996) studied PCDD/F persistence in a sludge-amended soil sample with presence of PCDD/Fs from 1968 to 1990. Half-lives for these PCDD/Fs in the sludge-amended soil after 1972 were of the order of 20 years (7300 days).

There is great variability in soil half-lives among the PCDD/F congeners between above studies. Soil half-life estimates for PCDD/Fs that currently have a toxicity equivalency factor (TEF) were given the greatest weight in determining a default soil half-life, where speciation of PCDD/Fs has been performed on facility emissions, unless only total PCDD/Fs are available (OEHHA 2003). Table G-4 shows the PCDD/F half-life results from the study (Kjeller and Rappe, 1995) found in the literature which speciated PCDD/F congeners.

Table G-4 Soil half-lives (days) for PCDD/Fs relevant to the “Hot Spots” program

Compound	TEF _{WHO-97}	Half-life (days) from Kjeller and Rappe (1995)
PCDDs		
2378-TCDD	1	37,500
12378-PeCDD	1	42,000
123478-HxCDD	0.1	100,000
123789-HxCDD	0.1	29,200
123678-HxCDD	0.1	23,000
1234678-HpCDD	0.01	37,500
12346789-OCDD	0.0001	54,200
PCDFs		
2378-TCDF	0.1	23,000
12378-PeCDF	0.05	18,750
23478-PeCDF	0.5	23,000
123478-HxCDF	0.1	25,000
123789-HxCDF	0.1	20,800
123678-HxCDF	0.1	29,200
234678-HxCDF	0.1	18,750
1234678-HpCDF	0.01	14,600
1234789-HpCDF	0.01	12,500
12346789-OCDF	0.0001	10,400
Arithmetic mean half-lives		30,600

The arithmetic mean of the suggested values from ten studies (6,986 days) cited above is recommended as the estimated soil half-life of TCDD/Fs if the facility is reporting emissions of total dioxins and furans.

G.2.8 Summary

The chemical-specific soil half-lives for each chemical are summarized as Table G-5 below.

TableG-5. Summary of Soil Half-life Values (Days).

Compound	Soil Half-life (days)
Arsenic	1.0 E+08
Beryllium	1.0 E+08
Cadmium	1.0 E+08
Chromium	1.0 E+08
Diethylhexylphthalate	1.5 E+01
Hexachlorobenzene	1.0 E+08
Hexachlorocyclohexanes	9.4 E+01
Lead	1.0 E+08
Mercury	1.0 E+08
4,4'-methylenedianiline	4.6 E+02
PAHs	4.3 E+02
PCBs	3.2 E+03
PCDD/F	7.0 E+03

For a chemical with individual congeners, such as PCBs, PAHs, PCDD/Fs, only the grand average was presented in Table G-5. When speciation of these chemicals has been performed on facility emissions, soil half-life data for individual congeners are summarized in Table G-2 (PCBs), Table G-3 (PAHs) and Table G-4 (PCDD/Fs).

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Appendix H

Root Uptake Factors

H.1 Introduction

Root uptake factors for crops have been estimated for toxic metals in the “Hot Spots” program. These toxic metals are subject to soil deposition and subsequent uptake by the roots of home raised produce. A root uptake factor is necessary to estimate a concentration in the plant from the concentration in the soil. An estimate of produce consumption can be applied to estimate dose to the residential receptor (Chapter 7). The soil-to-plant uptake factor (UF) is the ratio of the fresh weight contaminant concentration in the edible plant or plant part over the total concentration of the contaminant in wet weight soil:

$$UF = C_{f.w.plant} / C_{wet.w. soil} \quad (Eq. H-1)$$

where: $C_{f.w.plant}$ = fresh weight concentration in the plant (mg/kg)
 $C_{wet.w. soil}$ = wet weight concentration in soil (mg/kg)

In the last 25 years, a large number of studies have been published that investigated metal concentrations in edible plants grown in contaminated soils. Although most of these studies did not calculate the UF, data were often presented from which a UF could be calculated. OEHHA assembled the data from these studies into a database from which basic statistical analyses for chemical UFs were determined. The volume of studies that could be included in the database is quite large for some inorganic metals, with new studies frequently published. Our database is not an exhaustive compilation of all plant uptake studies published, however, enough data was found to reasonably estimate default UFs for most of the toxic metals and metalloids of concern.

The UFs calculated by OEHHA are based on the total metal concentration in soil and reflect the fact that most crop uptake studies estimate total metal soil concentration, usually by extraction with strong or moderately strong acids (e.g., 4 N sulfuric acid). A smaller body of uptake studies use various mild soil extraction processes (e.g., extraction with diethyltriaminopentaacetic acid) to try and estimate plant bioaccessible metal concentrations in soil. Once more studies become available using an established method for estimating bioaccessible metals in contaminated soil, OEHHA may also consider developing an algorithm that incorporates a bioaccessible metal uptake factor.

The ability for crops to accumulate and translocate toxic inorganic metals and metalloids to edible parts depends to a large extent on soil and climatic factors, plant genotype and agronomic management (McLaughlin et al., 1999). In order to be most applicable to Hot Spots risk analysis, a set of criteria was applied for the selection of data used in developing soil-to-plant uptake factors.

Data used to determine root uptake factors were limited to studies that estimated contaminant concentrations in edible portions of crops raised and harvested at maturity for human consumption. Crops that are commonly grown in backyard gardens in California were considered most relevant. For example, plant uptake studies in crops grown in tropical climates were not included in the database. Grain crops such as

wheat and rice were also not included in the database because these crops are unlikely to be grown in backyard gardens. In most field studies background soil contaminant levels were unknown or not presented. However, field studies were included in the database if the study indicated that the soil was contaminated due to human causes, or that the soil contaminant concentration was considered above background levels.

Another data selection factor was soil pH because soil pH is a major influence on root uptake. Most agricultural soils in California are near neutral, with a geometric mean pH=7.2 (Holmgren et al., 1993). The range of pHs for most agricultural soils in California are roughly estimated at between 5.5 and 7.6. Thus, plant uptake studies that investigated soils with pH values within this range were considered most useful for estimating soil-to-crop uptake factors. Acidic soils tend to increase the bioavailability of divalent cationic metals such as cadmium, lead, and mercury. UFs based on acidic soils may overestimate metal uptake from pH neutral soils.

A distinction is made in the database for contaminant source between freshly added inorganic salts and other forms of the chemicals. In general, fresh addition of metal salts to soil in laboratory experiments will represent the most available form of the metal to plants. UFs developed from these studies likely represent an upper limit for plant accumulation. Where possible, UFs were calculated based on field studies that estimated plant uptake due to human-caused contamination of soils. These sources primarily included mine waste, smelter deposits, vehicle and other urban emissions, other industrial sources, wastewater effluent, compost, fertilizer, dredged material, sewage sludge, fly ash and flue dust. Ideally, UFs would be based on airborne deposition of contaminants due to emissions from nearby industrial facilities. However, uptake data from these sources were often very limited.

Most of the plant uptake studies summarized in the database presented their contaminant concentration results on a dry weight basis for both the plants and the soil. However, the soil-to-plant UF in Eq. 7.6 (Chapter 7) is expressed as a ratio of fresh weight crop concentration per wet weight soil concentration. To adjust the soil-to-plant UFs to a fresh weight crop basis, dry-to-wet weight fractions of edible portions of crops were applied using literature sources containing water content data of raw fruits and vegetables (Watt and Merrill, 1975; Baes et al., 1984; USDA, 2009). A default value of 0.8 was applied to all UFs for the dry-to-wet weight adjustment of soil, unless water content data of soil was presented in the study (Clement Associates, 1988).

As a result, two types of soil-to-plant UFs can be generated for each metal contaminant: one based on the dry weight plant over dry weight soil, and the other based on fresh weight plant over wet weight soil. A UF based on dry weights of plant and soil may be beneficial because the ratio avoids the naturally wide variations in water content of the crops and the soil. On the other hand, estimates of fruit and vegetable consumption are based on fresh weight values for the crops, which were grown in irrigated soils. This type of UF is most applicable for contaminant exposure via the crop consumption pathway (Eq. 7.6).

Finally, some studies also presented uptake data for reference soils. This information was also entered into the database to estimate crop uptake based on control soils as well as crop uptake specifically due to deposited contaminants (i.e., contaminated soil minus control soil metal concentration). Metals of concern naturally present in soils may be largely present in the mineral fraction of the soil and not available for uptake by plants. However, it may be beneficial to know what the background soil-to-plant UF is for toxic metals to estimate the impact of anthropogenic sources of the same metals is on the soils and plants.

The database of the studies used in the analysis is presented at the end of this appendix. Studies were grouped according to each metal/metalloid for comparison purposes.

H.2 Arsenic

Arsenic can be present in well-drained soils as $\text{H}_2\text{AsO}_4^{-1}$ if the soil is acidic or as HAsO_4^{-2} if the soil is alkaline (Bhumbla and Keefer, 1994). Arsenite (As(III)) the reduced state of inorganic arsenic, is a toxic pollutant in natural environments. It is much more toxic and more soluble and mobile in soil than the oxidized state of inorganic arsenic, arsenate (As(V)). Under flooded conditions, As(III) would dominate, whereas aerobic conditions would favor the oxidation of As(III) to As(V). Arsenic accumulates in roots of plants grown on soils contaminated by arsenic pesticides. However, arsenic is not readily translocated to above-ground parts.

Although background mean levels of arsenic in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of arsenic in European and Canadian agricultural soils to be in the range of 5 to 12 mg/kg dry soil. Kloeke et al. (1984) reports that the range of arsenic in arable land to be 0.1 to 20 mg/kg dry soil. The typical dry weight concentration of arsenic in plants has been listed as 0.1 to 5 mg/kg (Vecera et al., 1999). In this document, all crops grown in As-polluted soils had an overall average dry weight arsenic concentration of about 2.5 mg/kg, which is within the range of typical plant concentrations.

Table H.1 Distribution Parameters for Arsenic Fresh Weight Soil-to-plant Uptake Factors

	Leafy	Exposed	Protected	Root
n	27	22	8	17
minimum	0.000275	0.0000538	0.000115	0.000338
maximum	0.055	0.132	0.27	0.045
mean	0.00983	0.0158	0.066	0.00828
median	0.00531	0.00138	0.032	0.00399
90 th percentile	0.0257	0.0403	0.19	0.0236
95 th percentile	0.0481	0.0674	0.23	0.0361

It was observed that lower UFs were recorded in plants growing in high As-polluted soils compared to plants growing in low-level As-polluted soils. This finding, in part, led to

the large range in UF values shown in Table H.1 for some types of crops. For example, in soils with low-level As contamination of < 12 mg/kg, a UF of 0.01 was calculated for both exposed and leafy crops. In exposed and leafy crops grown in soils with >12 to 745 mg/kg As (mean: 343 mg/kg), calculated UFs were 0.0002 and 0.002, respectively. This seems to suggest that many crops have the ability to resist uptake, or have a high excretion rate, of excessive amounts of As in highly polluted soils. The crop UFs in Table H.1 are based on the arithmetic mean value for low- to high-level As polluted soils.

H.2 Beryllium

Very little data could be found regarding plant uptake of beryllium from the soil. Measurable amounts of beryllium in plants is rarely observed and the toxicity of this metal to plants is reported to be high (Shacklette et al., 1978; Baes et al., 1984). Klope et al. (1984) estimates that a general dry weight plant/soil transfer coefficient for Be is in the range of 0.01 - 0.1, similar to that found for lead and mercury.

Single soil-to-plant data points from Baes et al. (1984) for leafy and protected crops were used in Table 7-6 to represent these particular crop types. These were the only UFs that could be located in the literature. Due to expected similarities in soil-to-plant transfer, the lead UFs for root and exposed crops were used to represent the root and exposed UFs for beryllium.

H.3 Cadmium

Cadmium has the most extensive literature on root uptake of any of the toxic metals. Compared to Pb, Cd is readily taken up by plants, but unlike the other heavy metals, Cd is not phytotoxic at low plant concentrations that pose a concern to human health (McLaughlin et al., 1999). Cadmium exists in solution mostly as the divalent cation, Cd^{2+} . Plant uptake of Cd is governed by a number of factors that include soil pH, organic matter, cation exchange capacity, clay type and amount, hydrous metal oxides, carbonates, and other inorganic compounds (Mahler et al., 1987; McLaughlin et al., 1996). Acidic soils, and soils with lower clay and humus content will increase availability of Cd to plants.

The mean concentration of Cd in uncontaminated U.S. agricultural soils is 0.27 mg/kg d.w., with 5th and 95th percentiles of 0.036 and 0.78 mg/kg d.w., respectively (Holmgren et al., 1993). The mean concentration of Cd for field-contaminated soils reviewed in this document was about 8 to 9 mg/kg d.w., with a range of 0.16 to 106.5 mg/kg d.w. Typical dry weight levels of Cd in plants are expected to be between 0.1 and 1 mg/kg (Vecera et al., 1999). In this document, the overall Cd concentration in crops grown in Cd-polluted soil was about 6 mg/kg.

Figure H.1. Cumulative distribution of the leafy crop UFs for cadmium from field studies in the literature (n=73, skewness = 3.05, kurtosis = 9.09)

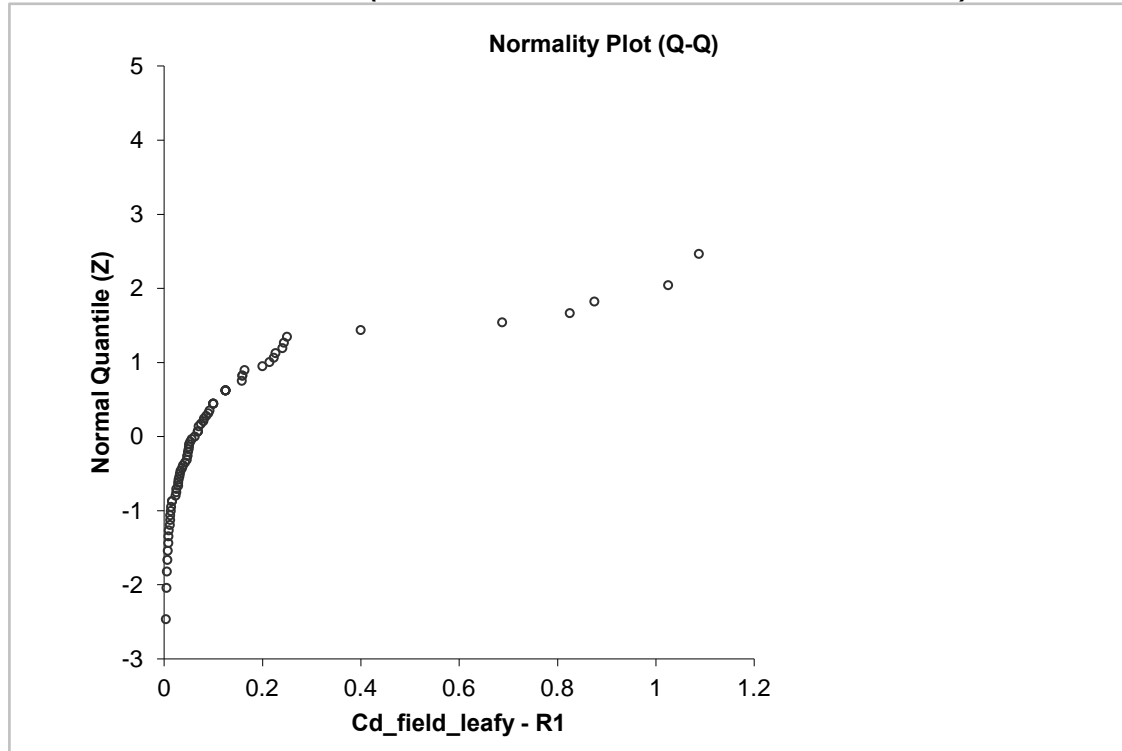


Table H.2 presents the UF distributions from field data only. UFs calculated from laboratory studies in which Cd salts were added to soils were not included in Table H.2, although there are a considerable number of these types of studies. Comparison of UFs calculated from field and Cd salt studies showed significantly greater UFs were obtained in crops grown in Cd salt-contaminated soil. For example, the mean leafy UF from Cd salt studies was 0.5 (n=27), which was significantly greater ($p < 0.0001$) than the leafy UF of 0.1 based on field studies (Table H.2). The field studies were chosen to calculate the UFs because they are likely more relevant for “Hot Spots” facility soil contamination.

Table H.2: Percentile Distribution for Cadmium Fresh Weight Soil-to-plant Uptake Factors

	Leafy	Exposed	Protected	Root
n	81	41	27	62
minimum	0.00375	0.0001	0.0002	0.00113
maximum	1.09	0.148	0.0688	0.913
mean	0.139	0.0216	0.0134	0.0683
median	0.0688	0.008	0.0064	0.0244
90 th percentile	0.244	0.0541	0.0294	0.124
95 th percentile	0.688	0.0863	0.0552	0.172

H.4 Chromium VI

Exposure to hexavalent chromium (Cr(VI)) as a contaminant in soil has been a contentious and complex risk assessment issue that has never been satisfactorily resolved. In both industrial and environmental situations Cr(III) and Cr(VI) can inter-convert, with reduction of Cr(VI) to Cr(III) generally being favored in most soils and sediments. Rapid oxidation of a portion of Cr(III) salts or hydroxides added to almost any soil with a pH above 5 was found to occur readily, provided the soil sample was fresh and kept moist and directly from the field (Bartlett and James, 1988). However, oxidation of Cr(III) to Cr(VI) in field soils is slow compared to well mixed soils in laboratory studies, and given opportunities for its reduction, accumulated Cr(VI) from inorganic sources may rarely be measurable.

Cr(VI) added to soils may be reduced, or absorbed, or may remain in solution depending on the organic matter content, pH, and texture of the soil (Cary, 1982). In neutral to basic soil, chromium will be more available to growing plants than in acidic soil probably due to the increased stability and presence of Cr(VI) in the basic pH range. For example, when Cr(VI) was added to near-neutral pH soil (6.65) under field conditions, most of the Cr(VI) was extracted from the soil unchanged three weeks later (Bloomfield and Pruden, 1980). Under the same field conditions, most of the added Cr(VI) to an acidic soil (pH 4.20) was reduced three weeks later. These results suggest that in some neutral pH agricultural soils, such as those found in California, constant deposition of Cr(VI) may result in accumulation of Cr(VI) in the soil and ground water.

As a soluble anion, Cr(VI) readily penetrates cell membranes, whereas Cr(III) is soluble at biological pHs only when organically complexed in low molecular weight organic complexes and, therefore, soil forms probably do not penetrate membranes (Bartlett and James, 1988). The difficulty for risk assessors is attempting to estimate what proportion of chromium deposited as Cr(VI) to soil will be available for plant uptake, presumably as Cr(VI). This problem is compounded by the difficulty of estimating the actual speciation of chromium in biological tissues during analysis. As a result, most studies only measure total chromium contents of plant parts.

Cr(III) in soil probably does not penetrate plant cell membranes as such, but is thought to undergo enhanced solubility in soil due to organic acids exuded by roots (James and Bartlett, 1984; Bartlett and James, 1988). This in turn leads to an increased oxidation of Cr(III) to Cr(VI) by soil manganese oxides. The oxidation of Cr(III) to anionic Cr(VI) enables its absorption by the roots. However, once absorbed by root tissues, it appears that most of the Cr(VI) is reduced again to Cr(III) and retained by the roots in a tightly bound or insoluble form or in a soluble complex (e.g., trioxalato chromate(III)) that is not translocated to the above-ground plant parts.

Evidence for the low translocation of chromium from roots has been observed by Lahouti (1979), in which crops accumulated chromium from nutrient solutions labeled with either $^{51}\text{Cr(III)}$ or $^{51}\text{Cr(VI)}$ retained about 98% of the elements in the roots. Of nine species of crops examined, the roots supplied with $^{51}\text{Cr(III)}$ contained more chromium than those supplied with $^{51}\text{Cr(VI)}$, but chromium added as $^{51}\text{Cr(VI)}$ was slightly better translocated to the shoots. In another study, onion plants were grown in soil after equivalent doses (total dose not provided) of either Cr(III) or Cr(VI) added to the soil (Srivastava et al., 1994). At the lower levels that did not injure the onion plants, the chromium concentration in the plants with Cr(VI) added to soil was only marginally higher than those with Cr(III) added to soil, with most of the chromium retained in the roots and bulb.

This finding seems to suggest that much of the chromium, either added as Cr(VI) or Cr(III), had reached an equilibrium in the soil prior to uptake by the roots. Field studies in which soils were contaminated by anthropogenic sources of Cr(VI) were difficult to come by. Soils contaminated with chromium, generally from sewage sludge, tannery waste, inorganic native chromium in mine waste, are mainly present as Cr(III). Often, the contaminated soils did not exhibit concentrations above the range of typical soil chromium levels of 2 to 50 mg/kg (Kloke et al., 1984), and no chromium control level was provided in the study. Quantitative data for plant uptake of chromium added as Cr(VI) in greenhouse studies are also limited. Cary et al. (1977a), (1977b) added Cr(VI) as K_2CrO_4 to soil over the first 29-40 days after seeding several crop varieties in pots, and then harvested the crops at maturity 70-110 days after seeding. From this data, leafy, exposed and protected crop UFs for total chromium were estimated (Table H.3). For the root UF, it was observed that roughly 10% of the chromium added as Cr(VI) to soil was incorporated in the above-ground plant parts, with the remainder incorporated into roots and bulbs (Srivastava et al., 1994). The difference between above-ground and root chromium was also reflected by a 10-fold greater concentration of chromium in roots compared to above-ground plant parts. Thus, the root UF is 10-fold greater than the leafy UF. It is currently unknown what proportion of chromium as Cr(VI) will be found in edible crops following absorption and translocation from the roots (Cary, 1982; Kimbrough et al., 1999). Bartlett and James (1988) surmised that if Cr(III) were to be translocated to above-ground plant parts, it is not unreasonable to think that if it enters the chloroplasts it might be oxidized to Cr(VI) in the powerful oxidative environment within the chloroplasts where water is oxidized to O_2^- . Skeffington (1976) showed 0.5% of the Cr(III) mixed with ground fresh barley roots was oxidized to Cr(VI). This data would suggest that a fraction of the chromium in roots is present as Cr(VI). Until further

characterization of the form of chromium found in edible crops is determined, the health protective assumption is that the chromium found in crops due to root uptake is in the form of Cr(VI).

Table H.3: Crop uptake factors for total chromium, added originally as chromium(VI) to the soil^a

	Leafy	Exposed	Protected	Root
N	3	1	3	- ^b
Minimum	0.18	-	0.0034	-
Maximum	0.42	-	0.19	-
Mean	0.3	0.02	0.07	3

^a Data was too limited to determine percentiles.

^b No quantitative data could be found for a root UF. The general finding that root levels of chromium are 10-fold greater than above-ground plant parts was to devise a root UF.

H.5 Fluoride

Fluoride (F) is strongly sorbed to soil when added as a salt, much stronger than other halide salts of iodine, bromine and chlorine (Sheppard et al., 1993). The generally low soluble F in most soils coupled with the fact that the root endodermis acts as a barrier means that transport from root to shoot will be limited (Davison, 1982). The lack of soil-to-plant field data for fluoride resulted in a reliance on laboratory studies which added fluoride salts to the soils. The resulting UFs are shown in Table H.4.

The most important F exposure route for plants is uptake via airborne deposition of soluble fluorides of HF and particulate fluoride salts on leaf surfaces. Fluoride that deposits on leaf surfaces can be taken up through stomata of leaves once it deposits on the surface. Uptake of F into plant leaves occurs by passive permeation of the undissociated HF molecule across the plasmalemma (Kronberger, 1987). Thus, HF behaves like a weak acid ($pK_a = 3.4$) when dissolved in water, where the ionic species becomes trapped within membrane-surrounded compartments after nonionic diffusion. Little fluoride moves downward in plants to roots, from leaf to leaf or from leaves to fruits. Assessing fluoride UFs for leafy crops near airborne industrial emissions of fluoride compounds may eventually require a different algorithm to estimate airborne fluoride accumulation in leafy crops.

Tea plants (*Camellia sinensis*) are known to accumulate high concentrations of F in their leaves from soil containing elevated levels of F, resulting in considerable amounts of F in tea beverages (Davison, 1983). However, it is not known if significant cultivation of tea plants occurs in California. There is also some evidence spinach can accumulate F from soil to a greater degree than other leafy crops (Kumpulainen and Koivistoinen, 1977). The maximum fluoride UF for leafy crops shown in Table H.4 is for spinach.

Table H.4: Fresh weight soil-to-plant uptake factors for fluoride^a

	Leafy	Exposed	Protected	Root
N	5	- ^b	1	2
Minimum	0.0006	-	-	0.003
Maximum	0.16	-	-	0.014
Mean	0.036	0.004	0.004	0.009

^a Data was too limited to determine percentiles.

^b No quantitative data could be found for an exposed crop UF, so the protected crop UF was used

H.6 Lead

Deposited lead (Pb) is strongly retained by most soils, resulting in lower plant concentrations (and lower UFs) relative to more bioaccessible metals such as cadmium and nickel (McLaughlin et al., 1999). Because of the usually low soil-to-root uptake, the above-ground plant parts are likely predominantly contaminated by airborne deposition of lead-containing dust or aerosols onto the plant surface (McBride, 1998). This finding emphasizes the importance of selecting studies in which the leafy plant samples are thoroughly washed prior to assessing root uptake and translocation of lead. Because inorganic lead most often exists as a divalent cation, maintaining alkaline soil conditions will reduce lead mobility in soil, while acidic soil conditions has been shown in some cases to increase soil mobility and uptake of lead through plant roots.

The mean concentration of Pb in uncontaminated U.S. agricultural soils is 12.3 mg/kg, with 5th and 95th percentiles of 4.0 and 23.0 mg/kg, respectively (Holmgren et al., 1993). The range of Pb concentrations in field-contaminated soils reviewed in this document was large, ranging from 11 mg/kg dry soil to nearly 5500 mg/kg dry soil. Typical dry weight concentrations of Pb in plants are reported to be 0.1 to 5 mg/kg (Vecera et al., 1999), whereas the overall average Pb concentration in crops grown in Pb-polluted soil reviewed in this document was about 9.5 mg/kg.

Table H.5: Percentile distribution for lead fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	77	38	24	57
minimum	0.0000375	0.00002	0.000075	0.0000425
maximum	0.0413	0.0475	0.0278	0.0375
mean	0.00770	0.00693	0.00282	0.00403
median	0.00298	0.00228	0.000912	0.00125
90 th percentile	0.0248	0.0214	0.00465	0.00962
95 th percentile	0.0308	0.0406	0.00711	0.015

H.7 Mercury

Determining the crop uptake of inorganic mercury (Hg) from soil can be problematic. (Caille et al., 2005) found that following application of radiolabeled $^{203}\text{HgCl}_2$ to sediment in a pot experiment, 33-73% of the leaf content in cabbage, rapeseed and pasture grass was due to volatilized Hg absorbed into the leaves. Presumably, the applied inorganic Hg^{2+} was emitted from the soil after reduction to Hg^0 in the soil whereupon it was absorbed by the leaves. Lindberg et al. (1979) observed the same phenomena in alfalfa grown in a chamber, in that above-ground plant parts primarily absorbed Hg vapor released from the soil originally contaminated with mercury mine waste including cinnabar (mercury(II) sulfide). However, the root levels of mercury were determined by direct uptake from contaminated soil and reflected the total Hg concentrations in the soil. Significantly, any Hg vapor emitted by a facility could also be absorbed directly onto leafy crops.

Nearly all studies examined by OEHHHA for crop Hg uptake from soil measured total Hg content and did not account for potential volatilization of elemental Hg from soil. Therefore, the soil-to-plant UF for mercury in above-ground plant parts (primarily leafy) includes both root uptake from soil and leaf uptake through volatilization from soil. It is unclear what portion of Hg oxidizes to inorganic Hg once absorbed by leaves, although mercury in food stuffs are mainly in the inorganic form (WHO, 1991). Therefore, a health protective assumption is that the Hg in crops is all in the inorganic form.

Another possible factor to consider is the uptake of methyl mercury (MeHg) by plants. Although it is not expected that Hot Spots facilities would emit MeHg, a fraction of total Hg emitted and deposited to soil could be converted to MeHg in soil. Generally, this may not be a concern in cropland soils, as the content of MeHg would be very low. Nevertheless, results by Gnamus et al. (2001) observed MeHg to be approximately 10 times more phytoavailable than total Hg in an ecotoxicology field study of an Hg-polluted region. Phytoavailability of both total Hg and MeHg increases with decreasing soil pH below 7 and decreased soil content of organic matter.

In rice paddies exposed to Hg smelting and mining facilities, it was found that the percent of total Hg in soil that was MeHg ranged from 0.092 to 0.003 percent (Horvat et al., 2003). However, the percent of total Hg that was MeHg in brown rice grown in the contaminated region ranged from 5 to 84 percent, indicating preferential uptake of MeHg from soil. The resulting UFs for rice ranged from 550 to 6000, suggesting rice may be a high accumulator of MeHg. However, the risk assessment conducted by Horvat et al. (2003) could not establish a clear correlation between total Hg and MeHg in soil and in rice, indicating that uptake and retention of Hg in rice is influenced by a number of factors other than total Hg in soil. Although background mean levels of Hg in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of Hg in European and Canadian agricultural soils to be in the range of 0.06 to 0.2 mg/kg dry soil. On average, the concentration of Hg in polluted soils reviewed in this document was about 3.6 mg/kg. Typical dry weight plant concentrations of Hg are listed as 0.001 to 0.3 mg/kg (Vecera et al., 1999). In this

document, the overall Hg concentration in crops grown in Hg-polluted soils was about 0.2 mg/kg.

<i>Table H.6: Percentile distribution for mercury fresh weight soil-to-plant uptake factors</i>	Leafy	Exposed	Protected	Root
n	33	23	15	18
minimum	0.00021	0.000248	0.000106	0.00111
maximum	0.0813	0.0938	0.0363	0.0588
mean	0.0163	0.00855	0.00804	0.0119
median	0.00875	0.00225	0.00514	0.00553
90th percentile	0.0478	0.0175	0.016	0.0274
95th percentile	0.06	0.0198	0.0223	0.0545

H.8 Nickel

Nickel (Ni) is considered to be one of the more mobile heavy metals in soils (Sauerbeck and Hein, 1991). However, in contrast to Cd, the toxicity of Ni in mammals is lower and phytotoxicity occurs at lower concentrations. Similar to other divalent, cationic metals, acidification of soil increases bioavailability, and liming of soil decreases bioavailability, of Ni to plants. The UF data presented in Table X.7 is based on field-contaminated studies. One study that added Ni salts to soil can be found in the database, but appeared to result in increased plant uptake compared to the field data and was, thus, not included for the UF calculations.

The mean concentration of Ni in uncontaminated U.S. agricultural soils is 23.9 mg/kg, with 5th and 95th percentiles of 4.1 and 56.8 mg/kg, respectively (Holmgren et al., 1993). The mean concentration of Ni for field-contaminated soils reviewed in this document was about 70 mg/kg d.w., with a range of 13 to 122 mg/kg d.w. Typical Ni levels in plants are expected to be in the range of 0.1 to 5 mg/kg dry weight (Vecera et al., 1999). In this report, the overall mean dry weight concentration of Ni in crops was about 9 mg/kg.

Table H.7: Percentile distribution for nickel fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	11	13	9	11
minimum	0.00135	0.00025	0.00875	0.00163
maximum	0.0375	0.00625	0.075	0.0175
mean	0.0145	0.00293	0.0305	0.00638
median	0.00888	0.00224	0.025	0.00463
90 th percentile	0.0250	0.00610	0.055	0.0125
95 th percentile	0.0313	0.00618	0.065	0.0150

H.9 Selenium

The major inorganic species of selenium (Se) in plant sources is selenate, which is translocated directly from the soil and is less readily bound to soil components than selenite (McLaughlin et al., 1999; Rayman, 2008). The more reduced forms, selenide and elemental Se, are virtually insoluble and do not contribute directly to plant uptake. Other major Se species in plants are biosynthesized, including selenomethionine, smaller amounts of selenocysteine, and Se-containing proteins. At pH values around 7.0 or greater, oxidation to the more soluble selenate ion is favored. Thus, endemic vegetation in alkaline, seleniferous soil of the western U.S. has evolved that is highly tolerant and can hyperaccumulate Se (McLaughlin et al., 1999).

However, potential Se-accumulators that are food sources for humans is largely limited to Brazil nuts, a tree crop that is not grown in California (Rayman et al., 2008). Crops of the Brassica (e.g., broccoli, cabbage) and Allium (e.g., onions, garlic, leeks, chives) families appear to more readily accumulate Se than other crops, and form the Se detoxification products Se-methyl-selenocysteine and gamma-glutamyl-Se-methyl-selenocysteine. Se-enriched plants have been shown in animals to have potent anti-tumor effects that are attributed to these Se detoxification products.

Though there is no direct evidence in humans, it is generally accepted on the basis of animal studies that inorganic forms of Se are more acutely toxic than organic species, selenite being slightly more toxic than selenate (Rayman et al., 2008). In chronic studies of humans, lower toxicity is seen with organically bound Se, although there are limited data on the toxicity of individual compounds.

Selenomethionine is known to be the main Se species present in the diet of Chinese who developed chronic selenosis from consumption of high-Se-containing maize and rice. Based on these Chinese studies, 1540 and 819 µg/day was established as the LOAEL and NOAEL, respectively, for total daily Se intake (Rayman, 2008). However, the levels found in crops rarely accumulate greater than 25-30 µg/g even in seleniferous areas suggesting other sources of Se are also contributors to chronic Se toxicity.

Although the UF data for Se was limited, an overall mean dry weight crop Se concentration of about 4 mg/kg was calculated from the reviewed studies, with a maximum crop concentration of 19 mg/kg. Kloeke et al. (1984) observed a general dry

weight UF for Se in plants would be 0.1 to 10. Based on the studies examined in this document, an overall dry weight uptake factor of 0.9 was calculated for crops grown in Se-polluted soils, which was within the range predicted. Field contamination studies were the primary source of the UF distribution data in Table X.8. The Se pollution sources included mainly fly ash, smelters and compost.

Table H.8: Percentile distribution for selenium fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	12	10	7	10
minimum	0.006	0.00132	0.00625	0.005
maximum	0.25	0.25	1.25	0.375
mean	0.0587	0.0415	0.256	0.0689
median	0.0328	0.0106	0.07	0.0195
90th percentile	0.12	0.104	0.678	0.15
95th percentile	0.179	0.177	0.964	0.263

H.10 Database

The database that lists all of the studies, values, with references is presented as Table H.9-1 through Table H.15-4 in the following pages.

Abbreviations in these tables:

soil conc bckd: the concentration of the chemical in the control soil samples

soil conc contam: the concentration of the chemical in the soil treated with the chemical

tissue conc bckg: the concentration of the chemical in the control tissue samples of the crop

tissue conc contam: the concentration of the chemical in the tissue of the crop grown in the soil treated with the chemical

contam: the related sample treated with the chemical

wt: weight

dw: dry weight

wet w: wet weight

ww: wet weight

Calculation:

$$\text{Uptake factor (contam) dry wt} = \frac{\text{tissue conc contam dry wt} - \text{tissue conc bckg dry wt}}{\text{soil conc contam} - \text{soil conc bckd}}$$

$$\text{Uptake factor (contam) wet wt plant/dw soil} = \text{Uptake factor (contam) dry wt} \times \text{dry-to-wet wt conversion factor}$$

$$\text{Uptake factor (contam) ww plant/wet w soil} = \frac{\text{Uptake factor (contam) wet wt plant/dw soil}}{\text{dry-to-wet weight fraction for soil (0.8)}}$$

Table H.9-1 Arsenic field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conver- sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Field		377	leaf mustard		20	0.05305	0.08	0.004244	0.005305	Clemente et al. (2005)
25% mine waste - greenhouse	23.3	187	lettuce	5.47	21.5	0.11497	0.045	0.005	0.00625	Cobb et al., (2000)
field-fly ash - pot	8.8	9.5	cabbage	0.2	0.3	0.03	0.08	0.003	0.00375	Furr et al. (1978a)
Field		6.04	Chinese cabbage			0.025	0.08	0.002	0.0025	Huang et al. (2006)
Field		6.04	leaf mustard			0.07125	0.08	0.0057	0.007125	Huang et al. 2006
Field		6.04	lettuce			0.046	0.05	0.0023	0.002875	Huang et al. 2006
Field		6.04	pakchoi			0.04625	0.08	0.0037	0.004625	Huang et al. 2006
Field		6.04	water spinach			0.07375	0.08	0.0059	0.007375	Huang et al. 2006
Field			amaranthus			0.55	0.08	0.044	0.055	Huq and Naidu (2005)
Field			cabbage			0.44	0.08	0.0352	0.044	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	kale	0.078	0.1	0.0056	0.08	0.00045	0.000563	Larsen et al., (1992)
wood preserve. Factory-field	3.4	17.9	lettuce	0.048	0.086	0.0048	0.05	0.00024	0.0003	Larsen et al., 1992
mining, smelting-field		446.64	cabbage		1.48	0.0033	0.08	0.00027	0.000338	Li et al., (2006)
mining, smelting-field		446.64	cabbage		1.21	0.0027	0.08	0.00022	0.000275	Li et al., 2006
mining, smelting-field		446.64	Chinese cabbage		1.85	0.0041	0.08	0.00034	0.000425	Li et al., 2006
mining, smelting-field		446.64	spinach		1.37	0.0031	0.08	0.00025	0.000313	Li et al., 2006
Field		6.01	amaranth		0.67	0.11148	0.08	0.008918	0.011148	Liu et al. (2006)
Field		6.01	cabbage		0.81	0.13478	0.08	0.010782	0.013478	Liu et al. 2006
Field		6.01	celery		0.49	0.08153	0.08	0.006522	0.008153	Liu et al. 2006
Field		6.01	Chinese cabbage		0.45	0.07488	0.08	0.00599	0.007488	Liu et al. 2006
Field		6.01	Chinese chive		0.57	0.09484	0.08	0.007587	0.009484	Liu et al. 2006
Field		5.54	leek		0.62	0.11191	0.08	0.008953	0.011191	Liu et al. 2006
field		6.01	pakchoi		3	0.49917	0.08	0.039933	0.049917	Liu et al. 2006
pot	9.83	745	Radish	0.28	14.4	0.01933	0.08	0.001546	0.001933	Mathe-Gaspar and Anton (2002)

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pot	9.83	745	Radish	0	48.7	0.06537	0.08	0.00523	0.006537	Mathe-Gaspar and Anton 2002
Env polluted soil - field		118	lettuce		7.2	0.06102	0.049	0.003	0.00375	Mattina et al., (2003)
Env polluted soil - field		125.9	spinach		1.55	0.012	0.093	0.0011	0.001375	Mattina et al., 2003

Average Arsenic uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00666 ± 0.00982

Table H.9-2 Arsenic field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash - pot	8.8	9.5	tomato	0.03	0.1	0.01	0.059	0.0006	0.00075	Furr et al. 1978
field		6.04	bottle gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field		6.04	cauliflower			0.00873	0.126	0.0011	0.001375	Huang et al. 2006
field		6.04	celery			0.05873	0.126	0.0074	0.00925	Huang et al. 2006
field		6.04	cowpea			0.00272	0.257	0.0007	0.000875	Huang et al. 2006
field		6.04	eggplant			0.00822	0.073	0.0006	0.00075	Huang et al. 2006
field		6.04	onion			0.0088	0.125	0.0011	0.001375	Huang et al. 2006
field		6.04	towel gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field			bean			0.27	0.111	0.02997	0.037463	Huq and Naidu 2005
field			cauliflower			0.84	0.126	0.10584	0.1323	Huq and Naidu 2005
field			tomato			0.55	0.059	0.03245	0.040563	Huq and Naidu 2005
mining, smelting-field		446.64	capsicum		0.75	0.0017	0.074	0.00013	0.000163	Li et al., 2006
mining, smelting-field		446.64	cucumber		0.49	0.0011	0.039	0.000043	5.38E-05	Li et al., 2006
mining, smelting-field		446.64	eggplant		0.45	0.001	0.073	0.000074	9.25E-05	Li et al., 2006
field		5.54	broccoli		0.59	0.1065	0.126	0.013419	0.016773	Liu et al. 2006

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		6.48	cucumber		0.53	0.08179	0.039	0.00319	0.003987	Liu et al. 2006
field		6.01	Eggplant		0.98	0.16306	0.073	0.011903	0.014879	Liu et al. 2006
field		6.01	kidney bean		2.98	0.49584	0.111	0.055038	0.068798	Liu et al. 2006
field		6.01	pepper		0.39	0.06489	0.126	0.008176	0.01022	Liu et al. 2006
field		6.01	tomato		0.46	0.07654	0.059	0.004516	0.005645	Liu et al. 2006
air dep, mine waste, poll. Water		459.02	capsicum		1.3		0.074	0.00021	0.000263	Liu et al., (2005)
air dep, mine waste, poll. Water	96.92	459.02	string bean	0.54	1.33	0.0029	0.111	0.00032	0.0004	Liu et al., 2005

Average Arsenic uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0158 ± 0.0313

Table H.9-3 Arsenic field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	dry-to- wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet t w soil	Reference
25% mine waste - greenhouse	23.3	187	bush bean	0.184	0.304	0.099	0.00016	0.0002	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	corn	0.1	0.2	0.895	0.02	0.025	Furr et al. 1978
field			cowpea			0.257	0.03341	0.041763	Huq and Naidu 2005
field			garlic			0.222	0.12654	0.158175	Huq and Naidu 2005
field			pea			0.257	0.21331	0.266638	Huq and Naidu 2005
field			pumpkin			0.222	0.03108	0.03885	Huq and Naidu 2005
mining, smelting-field		446.64	pumpkin		0.5	0.082	0.000092	0.000115	Li et al., 2006
air dep, mine waste, poll. Water		459.02	corn		0.21	0.261	0.00012	0.00015	Liu et al., 2005

Average Arsenic uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0664 ± 0.0962

Table H.9-4 Arsenic field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		13.3 (4-14)	potato		0.8	0.0602	0.222	0.013364	0.016706	Alam et al. (2003)
25% mine waste - greenhouse	23.3	187	radish	0.593	2.94	0.01572	0.047	0.00075	0.000938	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	carrot (peeled)	0.05	0.2	0.02	0.118	0.002	0.0025	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Onion (peeled)	0.1	0.3	0.03	0.125	0.004	0.005	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Potato (peeled)	0.1	0.1	0.01	0.222	0.002	0.0025	Furr et al. 1978
field		6.04	garlic			0.0245	0.2	0.0049	0.006125	Huang et al. 2006
field		6.04	radish			0.0285	0.2	0.0057	0.007125	Huang et al. 2006
field		6.04	taro			0.0165	0.2	0.0033	0.004125	Huang et al. 2006
field			carrot			0.23	0.118	0.02714	0.033925	Huq and Naidu 2005
field			radish			0.18	0.2	0.036	0.045	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	carrot (unpeeled)	0.032	0.042	0.0023	0.118	0.00027	0.000338	Larsen et al., 1992
wood preserve. Factory-field	3.4	17.9	potato (unpeeled)	0.037	0.077	0.0043	0.222	0.00095	0.001188	Larsen et al., 1992
field		5.54	carrot		0.15	0.02708	0.118	0.003195	0.003994	Liu et al. 2006
field		6.01	radish		0.22	0.03661	0.2	0.007321	0.009151	Liu et al. 2006
landfill-field		27	carrot (unpeeled)		0.17	0.0063	0.106	0.00067	0.000838	Samsøe-Petersen et al., (2002)
landfill-field		27	potato (unpeeled)		0.127	0.0047	0.094	0.00044	0.00055	Samsøe-Petersen et al., 2002
landfill-field		27	radish		0.27	0.01	0.059	0.00059	0.000738	Samsøe-Petersen et al., 2002

Average Arsenic uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00828 ± 0.0129

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	0.69	1.6	amaranth	0.81	3.85	2.406	0.08	0.1925	0.2406	Hu and Ding (2009)
field		0.16	amaranth		0.16	1.000	0.08	0.0800	0.1000	Liu et al. 2006
indust. Poll. Depo. - field		12	amaranthus		5.66	0.470	0.08	0.0380	0.0475	Pandey and Pandey, (2009)
Indust. sewage wastes - field	0.5	22	amaranthus	0.14	1.1	0.050	0.08	0.0040	0.0050	Srikanth et al., (1991)
field-wastewater	0.12	0.87	basil	0.16	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson (1986)
field		4.4	cabbage		0.3	0.068	0.08	0.0055	0.0068	Chumbley and Unwin (1982)
sewage sludge - pots		23.22	cabbage		1.77	0.076	0.08	0.0061	0.0076	Jackson & Alloway, (1991)
mining, smelting-field		7.43	cabbage		0.71	0.096	0.08	0.0077	0.0096	Li et al., 2006
mining, smelting-field		7.43	cabbage		1.29	0.170	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	cabbage		0.076	0.475	0.08	0.0380	0.0475	Liu et al. 2006
sewage sludge - field		10.5	cabbage		2.1	0.200	0.08	0.0200	0.0250	Muntau et al., (1987)
Indust. sewage wastes - field	0.5	22	cabbage	0.02	2.88	0.130	0.078	0.0100	0.0125	Srikanth et al., 1991
field - smelter	0.108	4.99	cabbage				0.052	0.1740	0.2175	Zheng et al. (2007a)a
field		1.6	celery		3.57	2.231	0.08	0.1785	0.2231	Hu and Ding 2009
field		0.16	celery		0.1	0.625	0.08	0.0500	0.0625	Liu et al. 2006
field - smelter	0.108	12.5	celery				0.058	0.1310	0.16375	Zheng et al. 2007a
mining, smelting-field		7.43	Chinese cabbage		1.31	0.180	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	Chinese cabbage		0.2	1.250	0.08	0.1000	0.1250	Liu et al. 2006
field		0.515	Chinese cabbage		0.2625	0.510	0.08	0.0408	0.0510	Wang et al. (2006)
field - smelter	0.108	22.8	Chinese cabbage				0.055	0.1280	0.16	Zheng et al. 2007a
field		0.16	Chinese chive		0.12	0.750	0.08	0.0600	0.0750	Liu et al. 2006
sewage sludge-field-grnhs		2.55	chinese leek		0.9	0.350	0.089	0.0310	0.0388	Yang et al., (2009)
field-wastewater	0.12	0.87	garden cress	0.1	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson 1986

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter	0.108	43.4	green onion				0.085	0.0440	0.055	Zheng et al. 2007a
field		0.17	leek		0.055	0.324	0.08	0.0259	0.0324	Liu et al. 2006
field - smelter	0.108	39.2	leek			2.250	0.08	0.1800	0.2250	Zheng et al. 2007a
field		7.8	lettuce		4.2	0.538	0.05	0.0269	0.0337	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	lettuce	1.61	5.37	0.890	0.045	0.0400	0.0500	Cobb et al., 2000
Env. contam. Soil 1a - potted		1.8	lettuce		2.5	1.400	0.049	0.0686	0.0858	Crews & Davies, (1985)
Env. contam. Soil 1b - potted		2.2	lettuce		7.8	3.500	0.049	0.1715	0.2144	Crews & Davies, 1985
Env. contam. Soil 2 - potted		4.5	lettuce		11.8	2.600	0.049	0.1274	0.1593	Crews & Davies, 1985
Env. contam. Soil 3 - potted		5.5	lettuce		20.5	3.700	0.049	0.1813	0.2266	Crews & Davies, 1985
field	0.69	1.6	lettuce	1.49	4.19	2.619	0.05	0.1309	0.1637	Hu and Ding 2009
fertilizer	0.53	0.6-0.86	lettuce				0.05	0.1950	0.2438	Huang et al. (2003)
fertilizer in field			lettuce				0.05	0.3199	0.3998	Huang et al. (2004)
sewage sludge - pots		23.22	lettuce		10.57	0.460	0.05	0.0230	0.0288	Jackson & Alloway, 1991
Env polluted soil - field		1	lettuce		2.6	2.600	0.049	0.1274	0.1593	Mattina et al., 2003
sewage sludge-field		2.2	lettuce		2.8	1.300	0.05	0.0650	0.0813	Preer et al., (1995)
smelter area - urban gardens	0.8	12.6	lettuce	0.41	7.55	0.600	0.049	0.0294	0.0368	Pruvot et al., (2006)
landfill-field		2.4	lettuce		0.552	0.230	0.05	0.0115	0.0144	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	lettuce		0.21	0.400	0.05	0.0200	0.0250	Samsoe-Petersen et al., 2002
fertilizer-field	ND	0.311	lettuce	ND	0.06	0.200	0.05	0.0100	0.0125	(Schroeder and Balassa, 1963)
fertilizer-field	ND	0.311	lettuce	ND	0.5	1.600	0.045	0.0720	0.0900	Schroeder & Balassa, 1963
urban gardens-field-to-grnhs	0.08	3.28	lettuce	0.65	1.73	0.760	0.045	0.0342	0.0428	Sterrett et al., (1996)
field - smelter	0.108	4.99	lettuce				0.042	0.2030	0.25375	Zheng et al. 2007
field-wastewater	0.12	0.87	mint	0.11	0.7	0.800	0.08	0.0640	0.0800	Shariatpanahi and Anderson 1986
field - smelter	0.108	20.1	mustard				0.071	0.0870	0.10875	Zheng et al. 2007
field		1.6	pakchoi		2.53	1.581	0.08	0.1265	0.1581	Hu and Ding 2009
field		0.16	pakchoi		0.11	0.688	0.08	0.0550	0.0688	Liu et al. 2006
field		0.515	Pakchoi		0.275	0.534	0.08	0.0427	0.0534	Wang et al. 2006
field		15.8	Pakchoi		0.21	0.090	0.08	0.0072	0.0090	Yan et al. (2007)
sewage sludge-field-		2.55	pakchoi		1.25	0.490	0.076	0.0370	0.0463	Yang et al., 2009

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
greenhouse										
field (industrial sewage irrigation)		2.69	palak (spinach)		1.5	0.560	0.08	0.0450	0.0563	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.26	palak (spinach)		2.1	0.930	0.08	0.0740	0.0925	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.8	palak (spinach)		2.85	1.000	0.08	0.0800	0.1000	Kumar Sharma et al., 2007
pot	0.167	30.5	Radish	0.388	8.78	0.288	0.08	0.0230	0.0288	Mathe-Gaspar and Anton 2002
pot	0.167	30.5	Radish	0.448	9.05	0.297	0.08	0.0237	0.0297	Mathe-Gaspar and Anton 2002
flooded gardens		1.31	sorrel		0.115	0.088	0.08	0.0070	0.0088	Sipter et al. (2008)
non-flooded gardens		0.43	sorrel		0.101	0.235	0.08	0.0188	0.0235	Sipter et al. 2008
field		4.6	spinach		4.6	1.000	0.08	0.0800	0.1000	Chumbley and Unwin 1982
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	1.48	2.18	8.300	0.08	0.6600	0.8250	He and Singh (1994)
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	2.32	2.85	10.860	0.08	0.8700	1.0875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	1.48	1.74	6.890	0.08	0.5500	0.6875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	2.32	2.58	10.210	0.08	0.8200	1.0250	He and Singh 1994
sewage sludge-field	0.48	5.32	spinach	0.94	12.76	1.991	0.08	0.1600	0.2000	Hooda et al., 1997
sewage sludge-field	1.6	4.3	spinach	0.01	0.14	0.030	0.08	0.0030	0.0038	Jamali et al., 2007
mining, smelting-field		7.43	spinach		1.06	0.140	0.08	0.0110	0.0138	Li et al., 2006
field (sewage-fed lake irrigation)			Spinach			2.500	0.08	0.2000	0.2500	Lokeshwari and Chandrappa 2006
Env polluted soil - field		0.7	spinach		5.3	7.600	0.093	0.7000	0.8750	Mattina et al., 2003
indust. Poll. Depo. - field		12	spinach		5.84	0.490	0.08	0.0390	0.0488	Pandey and Pandey, 2009
Indust. sewage wastes - field	0.5	22	spinach	0.13	6.4	0.290	0.086	0.0250	0.0313	Srikanth et al., 1991
field - smelter	0.108	43.4	spinach				0.088	0.0980	0.1225	Zheng et al. 2007
field		9.3	spring greens		1.1	0.118	0.08	0.0095	0.0118	Chumbley and Unwin 1982
sewage sludge - chamber	0.9	8.4	Swiss chard	2.2	11.2	1.300	0.08	0.1000	0.1250	Mahler et al., 1987
sewage sludge + limed - chamber	0.9	8.4	Swiss chard	1.7	8.4	1.000	0.08	0.0800	0.1000	Mahler et al., 1987

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
fertilizer-field greenhouse	0.07	1.13	Swiss chard	0.26	1.61	1.400	0.08	0.1000	0.1250	Mulla et al., (1980)
drilling fluid-greenhouse	0.6	19.4	swiss chard	1.5	26.9	1.400	0.08	0.1000	0.1250	Nelson et al., (1984)
sewage sludge-field		2.2	Swiss chard		3.15	1.400	0.08	0.1000	0.1250	Preer et al., 1995
field-wastewater	0.12	0.87	tarragon	0.14	0.05	0.060	0.08	0.0046	0.0058	Shariatpanahi and Anderson 1986
field		0.515	Water spinach		0.3625	0.704	0.08	0.0563	0.0704	Wang et al. 2006
field survey						0.507	0.08	0.0406	0.0507	Cambra et al. 1999

Average cadmium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.139 ± 0.214

Table H.10-2 Cadmium field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter	0.108	39.2	aubergine			0.513	0.081	0.0416	0.0519	Zheng et al. 2007a
indust. sewage-field-Egypt	ND	28	bell pepper		0.05	0.002	0.074	0.0001	0.0001	Gorbunov et al., 2003
field - smelter	0.108	20.1	bitter melon				0.066	0.0050	0.00625	Zheng et al. 2007a
landfill-field		2	blackberry					0.0025	0.0031	Samsøe-Petersen et al., 2002
field		0.17	broccoli		0.048	0.282	0.126	0.0356	0.0445	Liu et al. 2006
mining, smelting-field		7.43	capsicum		0.41	0.055	0.074	0.0040	0.0050	Li et al., 2006
air dep, mine waste, poll. Water		6.77	capsicum		1.37	0.200	0.074	0.0150	0.0188	Liu et al., 2005
field - smelter	0.108	39.2	capsicum			0.258	0.066	0.0170	0.0213	Zheng et al. 2007a
field		3.5	cauliflower		0.7	0.200	0.126	0.0252	0.0315	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	cucumber		0.06	0.002	0.039	0.0001	0.0001	Gorbunov et al., 2003
mining, smelting-field		7.43	cucumber		0.66	0.089	0.039	0.0035	0.0044	Li et al., 2006
field		0.16	cucumber		0.059	0.369	0.039	0.0144	0.0180	Liu et al. 2006

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge-field-grnhs		2.55	cucumber		0.2	0.080	0.04	0.0031	0.0039	Yang et al., 2009
mining, smelting-field		7.43	eggplant		0.4	0.054	0.073	0.0039	0.0049	Li et al., 2006
field		0.16	Eggplant		0.16	1.000	0.073	0.0730	0.0913	Liu et al. 2006
indust. Poll. Depo. - field		12	eggplant		4.18	0.350	0.073	0.0260	0.0325	Pandey and Pandey, 2009
field		0.515	Eggplant		0.3	0.638	0.073	0.0466	0.0583	Wang et al. 2006
indust. sewage-field-Egypt	ND	28	fig		0.015	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
sewage sludge-field	1.6	4.3	Indian squash	0.08	0.24	0.060	0.082	0.0050	0.0063	Jamali et al., (2007)
field		0.16	kidney bean		0.036	0.225	0.111	0.0250	0.0312	Liu et al. 2006
field-wastewater	0.12	0.87	leek	0.14	0.5	0.570	0.12	0.0690	0.0863	Shariatpanahi and Anderson 1986
indust. sewage-field-Egypt	ND	28	olive		0.03	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
landfill-field		2	pear					0.0034	0.0043	Samsoe-Petersen et al., 2002
sewage sludge-field			pepper				0.0408	0.0290	0.0362	Giordano et al., (1979)
field		0.16	pepper		0.15	0.938	0.126	0.1181	0.1477	Liu et al. 2006
field survey			peppers			0.053	0.126	0.0066	0.0083	Cambra et al. (1999)
landfill-field		2	plum					0.0006	0.0008	Samsoe-Petersen et al., 2002
sewage sludge-field			squash				0.082	0.0098	0.0123	Giordano et al., 1979
flooded gardens		1.31	squash		0.033	0.025	0.082	0.0021	0.0026	Sipter et al. 2008
non-flooded gardens		0.43	squash		0.005	0.012	0.082	0.0010	0.0012	Sipter et al. 2008
air dep, mine waste, poll. Water	2.08	6.77	string bean	0.21	0.67	0.099	0.111	0.0110	0.0138	Liu et al., 2005
25% mine waste - greenhouse	1.38	6.06	tomato	0.523	0.704	0.120	0.065	0.0078	0.0098	Cobb et al., 2000
field		0.15	tomato		0.11	0.733	0.059	0.0433	0.0541	Liu et al. 2006
indust. Poll. Depo. - field		12	tomato		4.96	0.410	0.059	0.0240	0.0300	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	tomato	0.15	1.23	0.098	0.065	0.0063	0.0079	Pruvot et al., 2006
flooded gardens		1.31	tomato		0.06	0.046	0.059	0.0027	0.0034	Sipter et al. 2008
non-flooded gardens		0.43	tomato		0.008	0.019	0.059	0.0011	0.0014	Sipter et al. 2008
smelter contam - field	0.08	4.4	tomato		0.43	0.098	0.065	0.0064	0.0080	Tomov & Alandjiyski, (2006)
sewage sludge-field-grnhs		2.55	tomato		0.2	0.080	0.033	0.0026	0.0033	Yang et al., 2009
field - smelter	0.11	43.4	tomato				0.056	0.0030	0.00375	Zheng et al. 2007a
field		0.515	Towel gourd		0.0976	0.189	0.082	0.0155	0.0194	Wang et al. 2006

Average cadmium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0216±0.0304

Table H.10-3 Cadmium field studies on protected Crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant /wet w soil	References
flooded gardens		1.31	bean		0.02	0.01527	0.111	0.0016947	0.0021	Sipter et al. 2008
non-flooded gardens		0.43	bean		0.01	0.02326	0.111	0.0025814	0.0032	Sipter et al. 2008
indust. sewage-field-Egypt	ND	28	bean (spot)		0.28	0.01	0.111	0.001	0.0013	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	28	bean (white)		0.26	0.009	0.111	0.001	0.0013	Gorbunov et al., 2003
sewage sludge-pot-field		4.6	beans		0.27	0.06	0.222	0.013	0.0163	Sauerbeck, 1991
field survey			broad beans			0.0108	0.126	0.0013608	0.0017	Cambra et al. 1999
25% mine waste - grhs	1.38	6.06	bush bean	0.145	0.01	0.0017	0.099	0.00017	0.0002	Cobb et al., 2000
sewage sludge-field			cantelope				0.06	0.0192	0.0240	Giordano et al., 1979
sewage sludge-field	1.6	4.3	cluster beans	0.04	0.2	0.05	0.111	0.005	0.0063	Jamali et al., 2007
field	0.26	25.3889	corn		0.2	0.00788	0.261	0.002056	0.0026	Bi et al. (2006)
air dep, mine waste, poll. Water		6.77	corn		0.47	0.069	0.261	0.018	0.0225	Liu et al., 2005
indust. sewage-field	0.072	3.72	corn	0.002	0.23	0.062	0.895	0.055	0.0688	Nan et al., (2002)
smelter area - ag field	0.4	8.1	corn	0.07	0.18	0.022	0.273	0.0062	0.0078	Pruvot et al., 2006
field		0.515	Cowpea		0.02724	0.05289	0.257	0.0135922	0.0170	Wang et al. 2006
field - smelter	0.108	43.4	cowpea				0.097	0.004	0.005	Zheng et al. 2007a
landfill-field		2	green bean		0.098	0.041	0.027	0.0011	0.0014	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	green bean		0.009	0.02	0.111	0.002	0.0025	Samsoe-Petersen et al., 2002
landfill-field		2	hazelnut					0.004	0.0050	Samsoe-Petersen et al., 2002
field - smelter	0.108	39.2	kidney bean			0.119	0.103	0.012257	0.0153	Zheng et al. 2007a
fertilizer-field	ND	0.311	onion	ND	0.024	0.08	0.125	0.01	0.0125	Schroeder & Balassa, 1963
fertilizer-field	ND	0.311	pea	ND	0.04	0.1	0.257	0.03	0.0375	Schroeder & Balassa, 1963
sewage sludge-field	1.6	4.3	peas	0.075	0.2	0.05	0.257	0.01	0.0125	Jamali et al., 2007
sewage sludge-pot-field		4.6	peas		0.2	0.04	0.257	0.01	0.0125	Sauerbeck, 1991
mining, smelting-field		7.43	pumpkin		0.46	0.062	0.082	0.0051	0.0064	Li et al., 2006
field - smelter	0.108	43.4	pumpkin				0.065	0.001	0.001	Zheng et al. 2007a
fertilizer-field	ND	0.311	string bean	ND	0.015	0.05	0.111	0.01	0.0125	Schroeder & Balassa, 1963

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant /wet w soil	References
field		7.8	sweet corn		1.5	0.19231	0.261	0.0501923	0.0627	Chumbley and Unwin 1982

Average cadmium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0134±0.0175

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
fertilizer-field	ND	0.311	beet	ND	0.045	0.100	0.2	0.0300	0.0375	Schroeder & Balassa, 1963
field		6.5	beetroot		2	0.308	0.222	0.0683	0.0854	Chumbley and Unwin 1982
smelter - field - home gardens		40.6	carrot		4.4	0.110	0.118	0.0130	0.0163	Chaney et al., (1988)
sewage sludge-field	0.48	5.32	carrot	0.63	1.71	0.350	0.118	0.0410	0.0513	Hooda et al., 1997
field		0.17	carrot		0.085	0.500	0.118	0.0590	0.0738	Liu et al. 2006
indust. Poll. Depo. - field		12	carrot		2.06	0.170	0.118	0.0200	0.0250	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	carrot	0.085	1.53	0.120	0.118	0.0140	0.0175	Pruvot et al., 2006
fertilizer-field	ND	0.311	carrot	ND	0.068	0.200	0.118	0.0300	0.0375	Schroeder & Balassa, 1963
flooded gardens		1.31	carrot		0.13	0.099	0.118	0.0117	0.0146	Sipter et al. 2008
non-flooded gardens		0.43	carrot		0.068	0.158	0.118	0.0187	0.0233	Sipter et al. 2008
contam-irrig. water - greenhouse		3.6	carrot		1.22	0.340	0.135	0.0460	0.0575	Zheng et al., (2008)
sewage sludge-field-greenhouse		2.55	carrot		0.7	0.270	0.11	0.0300	0.0375	Yang et al., 2009
field - smelter	0.108	39.2	carrot			0.752	0.088	0.0662	0.0827	Zheng et al. 2007a
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.115	0.145	0.550	0.118	0.0650	0.0813	He and Singh 1994
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.125	0.165	0.630	0.118	0.0740	0.0925	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.115	0.135	0.530	0.118	0.0630	0.0788	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.125	0.15	0.590	0.118	0.0700	0.0875	He and Singh 1994
fertilizers w/ Cd		0.3	carrot (unpeeled)		0.25	0.800	0.11	0.0900	0.1125	Jansson and Oborn, (2000)
landfill-field		2.4	carrot (unpeeled)		0.26	0.110	0.127	0.0140	0.0175	Samsøe-Petersen et al., 2002

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Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
moderate urban poll -field		0.56	carrot (unpeeled)		0.12	0.200	0.118	0.0300	0.0375	Samsøe-Petersen et al., 2002
sewage sludge-pot-field		4.6	carrots		0.9	0.200	0.118	0.0200	0.0250	Sauerbeck, 1991
field survey			chard			0.519	0.2	0.1038	0.1298	Cambra et al. 1999
indust. sewage-field-Egypt	ND	28	garlic		0.21	0.008	0.125	0.0009	0.0011	Gorbunov et al., 2003
smelter area - urban gardens	0.8	12.6	leek	0.14	1.58	0.130	0.146	0.0180	0.0225	Pruvot et al., 2006
field		3.1	leeks		0.8	0.258	0.2	0.0516	0.0645	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	onion		0.27	0.010	0.125	0.0010	0.0013	Gorbunov et al., 2003
field-wastewater	0.12	0.87	onion	0.12	0.3	0.340	0.125	0.0400	0.0500	Shariatpanahi and Anderson 1986
flooded gardens		1.31	onion		0.07	0.053	0.125	0.0067	0.0083	Sipter et al. 2008
non-flooded gardens		0.43	onion		0.056	0.130	0.125	0.0163	0.0203	Sipter et al. 2008
field survey			onions			0.105	0.125	0.0132	0.0164	Cambra et al. 1999
fertilizer-field	ND	0.311	parsnip	0.15	0.7	2.200	0.2	0.5000	0.6250	Schroeder & Balassa, 1963
smelter - field - home gardens		13.2	potato		3.6	0.270	0.202	0.7300	0.9125	Chaney et al., 1988
field		10.8	potato		0.6	0.056	0.222	0.0123	0.0154	Chumbley and Unwin 1982
smelter flue-dust	0.3	106.5	potato	0.16	1.67	0.016	0.222	0.0035	0.0044	Dudka et al. 1996
smelter flue-dust	0.3	54.4	potato	0.16	2.12	0.039	0.222	0.0087	0.0108	Dudka et al. 1996
smelter flue-dust	0.3	7.1	potato	0.16	0.53	0.075	0.222	0.0166	0.0207	Dudka et al. 1996
smelter flue-dust	0.3	3.2	potato	0.16	0.42	0.131	0.222	0.0291	0.0364	Dudka et al. 1996
smelter area - ag field	0.4	8.1	potato	0.3	0.45	0.056	0.202	0.0110	0.0138	Pruvot et al., 2006
smelter area - urban gardens	0.8	12.6	potato	0.05	0.54	0.043	0.202	0.0087	0.0109	Pruvot et al., 2006
fertilizer-field	ND	0.311	potato	ND	0.015	0.050	0.222	0.0100	0.0125	Schroeder & Balassa, 1963
smelter contam - field	0.08	4.4	potato		0.097	0.022	0.202	0.0044	0.0055	Tomov & Alandjiyski, 2006
sewage sludge - pots		23.22	potato (peeled)		0.3	0.013	0.222	0.0029	0.0036	Jackson & Alloway, 1991
sewage sludge-field		2.77	potato (peeled)		0.07	0.030	0.218	0.0055	0.0069	Smith (1994)
landfill-field		2.4	potato (unpeeled)		0.089	0.037	0.135	0.0050	0.0063	Samsøe-Petersen et al., 2002
moderate urban poll -field		0.56	potato(unpeeled)		0.05	0.090	0.222	0.0200	0.0250	Samsøe-Petersen et al., 2002
field		2.7	radish		1.7	0.630	0.222	0.1398	0.1747	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	radish	0.01	2.31	0.380	0.047	0.0180	0.0225	Cobb et al., 2000
indust. sewage-field-Egypt	ND	28	radish		0.28	0.010	0.085	0.0009	0.0011	Gorbunov et al., 2003
field		0.16	radish		0.083	0.519	0.2	0.1038	0.1297	Liu et al. 2006
field (sewage-fed lake irrigation)			Radish			1.600	0.2	0.3200	0.4000	Lokeshwari and Chandrappa 2006

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
indust. Poll. Depo. - field		12	radish		2.61	0.220	0.085	0.0190	0.0238	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	radish	0	2.12	0.170	0.047	0.0079	0.0099	Pruvot et al., 2006
landfill-field		2.4	radish		0.19	0.080	0.041	0.0033	0.0041	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	radish		0.071	0.100	0.085	0.0100	0.0125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		4.6	radish		1.1	0.200	0.05	0.0100	0.0125	Sauerbeck, 1991
fertilizer-field	ND	0.311	radish	ND	0.1	0.300	0.2	0.0600	0.0750	Schroeder & Balassa, 1963
field-wastewater	0.12	0.87	radish	0.18	0.45	0.520	0.085	0.0400	0.0500	Shariatpanahi and Anderson 1986
contam-irrig. water - greenhouse		3.6	radish		1.09	0.300	0.083	0.0250	0.0313	Zheng et al., 2008
sewage sludge-field-greenhouse		2.55	radish		0.5	0.200	0.05	0.0098	0.0123	Yang et al., 2009
field		4.8	salad onions		1	0.208	0.125	0.0260	0.0326	Chumbley and Unwin 1982
fertilizer-field	ND	0.311	turnip	ND	0.15	0.500	0.2	0.1000	0.1250	Schroeder & Balassa, 1963
field - smelter	0.108	39.2	turnip			0.027	0.108	0.0029	0.0036	Zheng et al. 2007a

Average cadmium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0683±0.144

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pots -env. chamber	30	300	cabbage		2.4	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
pots -env. chamber	30	300	rape		2.3	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
field		117	cabbage		0.3	0.0026	0.08	0.000205	0.0002564	Chumbley and Unwin 1982
field		155	lettuce		2.3	0.0148	0.05	0.000742	0.0009274	Chumbley and Unwin 1982
field		124	spinach		3.7	0.0298	0.08	0.002387	0.0029839	Chumbley and Unwin 1982
field		214	spring greens		2.3	0.0107	0.08	0.00086	0.0010748	Chumbley and Unwin 1982

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Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		532	leaf mustard		21	0.0395	0.08	0.003158	0.0039474	Clemente et al. 2005
25% mine waste - grnhs	60.9	3600	lettuce	29.8	227	0.0631	0.045	0.002838	0.0035469	Cobb et al., 2000
Env. contam. Soil 1a - potted - outside		301	lettuce		2	0.0066	0.049	0.000326	0.000407	Crews & Davies, 1985
Env. contam. Soil 1b - potted - outside		169	lettuce		7.7	0.0456	0.049	0.002233	0.0027907	Crews & Davies, 1985
Env. contam. Soil 2 - potted - outside		754	lettuce		5.7	0.0076	0.049	0.00037	0.000463	Crews & Davies, 1985
Env. contam. Soil 3 - potted - outside		850	lettuce		14.3	0.0168	0.049	0.000824	0.0010304	Crews & Davies, 1985
urban gardens-field			cilantro				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			collard greens				0.147	0.0004	0.0005	Finster et al., 2004
urban gardens-field			coriander				0.08	0.003	0.00375	Finster et al., 2004
urban gardens-field			ipasote				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			lemon balm				0.08	0.001	0.00125	Finster et al., 2004
urban gardens-field			mint				0.08	0.0009	0.001125	Finster et al., 2004
urban gardens-field			rhubarb				0.052	0.00047	0.0005875	Finster et al., 2004
urban gardens-field			Swiss chard				0.089	0.0027	0.003375	Finster et al., 2004
sewage sludge-field	70	259	spinach	0.82	0.95	0.0080	0.08	0.0006	0.00075	Hooda et al., 1997
field	65.9	361	amaranth	2.66	45.7	0.1266	0.08	0.010127	0.0126593	Hu and Ding 2009
field		361	celery		22.1	0.0612	0.08	0.004898	0.0061219	Hu and Ding 2009
field	65.9	361	lettuce	1.14	37.5	0.1039	0.05	0.005194	0.0064924	Hu and Ding 2009
field		361	pakchoi		36.2	0.1003	0.08	0.008022	0.0100277	Hu and Ding 2009
Pb arsenate - grnhs	60.9	342.3	lettuce	10.2	12.5	0.0400	0.05	0.002	0.0025	Hutchinson et al. 1974
sewage sludge-field	21.1	67.4	spinach	0.33	1.2	0.0200	0.08	0.001	0.00125	Jamali et al., 2007
mining, smelting-field		223.22	cabbage			0.0500	0.08	0.004	0.005	Li et al., 2006
mining, smelting-field		223.22	cabbage			0.0490	0.08	0.0039	0.004875	Li et al., 2006
mining, smelting-field		223.22	Chinese cabbage			0.0780	0.08	0.0062	0.00775	Li et al., 2006
mining, smelting-field		223.22	spinach			0.0700	0.08	0.0056	0.007	Li et al., 2006
field		14.48	amaranth		1.91	0.1319	0.08	0.010552	0.0131906	Liu et al. 2006
field		14.48	cabbage		1	0.0691	0.08	0.005525	0.0069061	Liu et al. 2006
field		14.48	celery		1.76	0.1215	0.08	0.009724	0.0121547	Liu et al. 2006

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Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		14.48	Chinese cabbage		2.05	0.1416	0.08	0.011326	0.0141575	Liu et al. 2006
field		14.48	Chinese chive		2.53	0.1747	0.08	0.013978	0.0174724	Liu et al. 2006
field		14.48	pakchoi		2.02	0.1395	0.08	0.01116	0.0139503	Liu et al. 2006
pot	18.5	2897	Radish	2.9	94.3	0.0326	0.047	0.00153	0.0019124	Mathe-Gaspar and Anton 2002
pot	18.5	2897	Radish	2.4	272.4	0.0940	0.047	0.004419	0.0055242	Mathe-Gaspar and Anton 2002
sewage sludge - field		775	cabbage		0.31	0.0004	0.08	0.00003	0.0000375	Muntau et al., 1987
drilling fluid-grnhs	17	1131	swiss chard	1.7	9.2	0.0080	0.08	0.0007	0.000875	Nelson et al., 1984
Env. contam. Soil (paint?) - potted - grnhs		2000	collard		8	0.0040	0.147	0.0006	0.00075	Nicklow et al., (1983)
Env. contam. Soil (paint?) - potted - grnhs		2000	kale		7	0.0035	0.173	0.0006	0.00075	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	lettuce		25	0.0125	0.049	0.000613	0.0007656	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	amaranthus		18.44	0.1100	0.08	0.0088	0.011	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	spinach		19.58	0.1200	0.08	0.0096	0.012	Pandey and Pandey, 2009
sewage sludge-field		98	lettuce			0.0200	0.05	0.001	0.00125	Preer et al., 1995
sewage sludge-field		98	Swiss chard			0.0300	0.08	0.003	0.00375	Preer et al., 1995
smelter area - urban gardens - field	84	872	lettuce	2.24	6.93	0.0079	0.049	0.000387	0.0004839	Pruvot et al., 2006
landfill-field		1000	lettuce		1.3	0.0013	0.05	0.000065	8.125E-05	Samsøe-Petersen et al., 2002
moderate urban poll -field		130	lettuce		0.25	0.0020	0.05	0.0001	0.000125	Samsøe-Petersen et al., 2002
field-wastewater	0.32	2.04	basil	0.18	0.84	0.4100	0.08	0.033	0.04125	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	garden cress	0.16	0.8	0.3900	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	mint	0.29	0.78	0.3800	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	tarragon	0.15	0.68	0.3300	0.08	0.027	0.03375	Shariatpanahi and Anderson 1986
flooded gardens		85.2	sorrel		0.99	0.0116	0.08	0.00093	0.001162	Sipter et al. 2008
non-flooded gardens		27.8	sorrel		0.295	0.0106	0.08	0.000849	0.0010612	Sipter et al. 2008
sewage sludge-field			spinach				0.08	0.00048	0.0006	Sridhara Chary et al., 2008
Indust. sewage wastes - field	3.4	183.5	amaranthus	0.12	12.2	0.0660	0.08	0.0054	0.00675	Srikanth et al., 1991
Indust. sewage wastes - field	3.4	183.5	cabbage	0.64	7.52	0.0410	0.078	0.0032	0.004	Srikanth et al., 1991

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Indust. sewage wastes - field	3.4	183.5	spinach	0.05	14.94	0.0810	0.086	0.007	0.00875	Srikanth et al., 1991
urban gardens-field-to-grnhs	12	1601	lettuce	2.22	8.67	0.0080	0.045	0.00036	0.00045	Sterrett et al., 1996
field		71.31	Chinese cabbage		0.65	0.0091	0.08	0.000729	0.0009115	Wang et al. 2006
field		71.31	Pakchoi		0.7625	0.0107	0.08	0.000855	0.0010693	Wang et al. 2006
field		71.31	Water spinach		1.2125	0.0170	0.08	0.00136	0.0017003	Wang et al. 2006
field		400.3	Pakchoi		3.28	0.0680	0.08	0.00544	0.0068	Yan et al. 2007
field - smelter	21.6	319.6	leek			0.2760	0.08	0.02208	0.0276	Zheng et al. 2007a
field - smelter		158	Chinese cabbage				0.055	0.018	0.023	Zheng et al. 2007b
field - smelter		297	green onion				0.085	0.006	0.008	Zheng et al. 2007b
field - smelter		297	spinach				0.088	0.025	0.03	Zheng et al. 2007b
field - smelter		139	celery				0.058	0.016	0.02	Zheng et al. 2007b
field - smelter		111	cabbage				0.052	0.019	0.024	Zheng et al. 2007b
field - smelter		111	lettuce				0.042	0.024	0.03	Zheng et al. 2007b
field - smelter		167	mustard				0.071	0.021	0.026	Zheng et al. 2007b

Average lead uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0077 ± 0.0104

Table H.11-2 Lead field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		12	peach		1.4	0.1167	0.131	0.015283	0.0191042	Basar and Aydmalp (2005)
field		12	peach		2.9	0.2417	0.131	0.031658	0.0395729	Basar and Aydmalp 2005
field		11	peach		0.8	0.0727	0.131	0.009527	0.0119091	Basar and Aydmalp 2005
field		137	cauliflower		2	0.0146	0.126	0.001839	0.0022993	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	334	bell pepper		0.4	0.0010	0.074	0.00007	0.0000875	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	cucumber		0.3	0.0009	0.039	0.00004	0.00005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	fig		0.6	0.0020	0.225	0.00045	0.0005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	olive		0.3	0.0009	0.2	0.0002	0.00025	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	Indian squash	0.33	1.4	0.0200	0.082	0.002	0.0025	Jamali et al., 2007
mining, smelting-field		223.22	capsicum			0.0370	0.074	0.0027	0.003375	Li et al., 2006
mining, smelting-field		223.22	cucumber			0.0460	0.039	0.0018	0.00225	Li et al., 2006
mining, smelting-field		223.22	eggplant			0.0220	0.073	0.0016	0.002	Li et al., 2006
field		14.49	broccoli		0.34	0.0235	0.126	0.002957	0.0036957	Liu et al. 2006
field		14.48	cucumber		1.39	0.0960	0.039	0.003744	0.0046797	Liu et al. 2006
field		14.48	Eggplant		1.3	0.0898	0.073	0.006554	0.0081923	Liu et al. 2006
field		14.48	kidney bean		0.91	0.0628	0.111	0.006976	0.0087198	Liu et al. 2006
field		14.48	pepper		4.25	0.2935	0.126	0.036982	0.0462276	Liu et al. 2006
field		14.47	tomato		5.23	0.3614	0.059	0.021325	0.026656	Liu et al. 2006
air dep, mine waste, poll. Water		751.98	capsicum		4.58	0.0061	0.074	0.00045	0.0005625	Liu et al., 2005
air dep, mine waste, poll. Water	60.49	751.98	string bean	0.84	5.82	0.0077	0.111	0.00086	0.001075	Liu et al., 2005
indust. Poll. Depo. - field		165.85	eggplant		13.15	0.0790	0.073	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	tomato		15.2	0.0920	0.059	0.0054	0.00675	Pandey and Pandey, 2009
smelter area - urban gardens - field	84	872	tomato	0	1.38	0.0016	0.065	0.0001	0.000125	Pruvot et al., 2006
Kalvebod area		613	blackberry					0.000026	0.0000325	Samsøe-Petersen et al., 2002
Kalvebod area		613	pear					0.000016	0.00002	Samsøe-Petersen et al., 2002
Kalvebod area		613	plum					0.000016	0.00002	Samsøe-Petersen et al., 2002
field-wastewater	0.32	2.04	leek	0.2	0.65	0.3200	0.12	0.038	0.0475	Shariatpanahi and Anderson 1986

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Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conver- sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
flooded gardens		85.2	squash		0.673	0.0079	0.082	0.000648	0.0008097	Sipter et al. 2008
flooded gardens		85.2	tomato		0.48	0.0056	0.059	0.000332	0.0004155	Sipter et al. 2008
non-flooded gardens		27.8	squash		0.079	0.0028	0.082	0.000233	0.0002913	Sipter et al. 2008
non-flooded gardens		27.8	tomato		0.083	0.0030	0.059	0.000176	0.0002202	Sipter et al. 2008
smelter contam - field	22	163	tomato		7.15	0.0440	0.065	0.0029	0.003625	Tomov & Alandjiyski, 2006
field		71.31	Eggplant		0.3973	0.0056	0.073	0.000407	0.0005083	Wang et al. 2006
field		71.31	Towel gourd		0.3415	0.0048	0.082	0.000393	0.0004908	Wang et al. 2006
field - smelter	21.6	319.6	aubergine			0.0240	0.066	0.001584	0.00198	Zheng et al. 2007a
field - smelter	21.6	319.6	capsicum			0.0240	0.081	0.001944	0.00243	Zheng et al. 2007a
field - smelter		297	tomato				0.056	0.002	0.003	Zheng et al. 2007b
field - smelter		167	bitter melon				0.066	0.003	0.004	Zheng et al. 2007b

Average lead uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00693±0.0124

Table H.11-3 Lead field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	50	318.056	corn		1.1	0.0035	0.261	0.000903	0.0011283	Bi et al. 2006
field		156	sweet corn		0.1	0.0006	0.261	0.000167	0.0002091	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	bush bean	5.53	0	-	0.099	0.00017	0.0002125	Cobb et al., 2000
indust. sewage-field-Egypt	ND	334	bean (spot)		2.2	0.0070	0.894	0.006	0.0075	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	bean (white)		0.9	0.0030	0.894	0.003	0.00375	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	cluster beans	0.104	0.6	0.0090	0.111	0.001	0.00125	Jamali et al., 2007
sewage sludge-field	21.1	67.4	peas	0.22	0.74	0.0100	0.257	0.003	0.00375	Jamali et al., 2007
mining, smelting-field		223.22	pumpkin			0.0470	0.082	0.0039	0.004875	Li et al., 2006
air dep, mine waste, poll. Water		751.98	corn		1.91	0.0025	0.261	0.00066	0.000825	Liu et al., 2005
field (sewage-fed lake irrigation)			Beans			0.2000	0.111	0.0222	0.02775	Lokeshwari and Chandrappa 2006
smelter area - ag field	30	440	corn	0	0.92	0.0021	0.273	0.00057	0.0007125	Pruvot et al., 2006
Kalvebod area		613	hazelnut					0.00073	0.0009125	Samsøe-Petersen et al., 2002
landfill-field		1000	green bean		1.4	0.0014	0.042	0.00006	0.000075	Samsøe-Petersen et al., 2002
moderate urban poll -field		130	green bean		0.18	0.0010	0.111	0.0002	0.00025	Samsøe-Petersen et al., 2002
sewage sludge-pot-field		154	beans			0.0080	0.222	0.002	0.0025	Sauerbeck, 1991
sewage sludge-pot-field		154	peas			0.0010	0.257	0.0003	0.000375	Sauerbeck, 1991
flooded gardens		85.2	bean		0.26	0.0031	0.111	0.000339	0.0004234	Sipter et al. 2008
non-flooded gardens		27.8	bean		0.141	0.0051	0.111	0.000563	0.0007037	Sipter et al. 2008
field		71.31	Cowpea		0.2023	0.0028	0.257	0.000729	0.0009115	Wang et al. 2006
field - smelter	21.6	319.6	kidney bean			0.0320	0.103	0.003296	0.00412	Zheng et al. 2007a
field - smelter		297	cowpea				0.097	0.003	0.004	Zheng et al. 2007b
field - smelter		297	pumpkin				0.065	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00282±0.00565

Table H.11-4 Lead field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		28	potato		0.5	0.0179	0.222	0.003974	0.0049673	Alam et al. 2003
salt	40.5	744.5	carrot	0.312	5.754	0.0077	0.118	0.000912	0.00114	Alexander et al. (2006)
salt	40.5	744.5	Onion	1.418	7.458	0.0100	0.125	0.001252	0.0015652	Alexander et al. 2006
smelter - field - home gardens		130	carrot		2.2	0.0169	0.118	0.002	0.0025	Chaney et al., 1988
smelter - field - home gardens		48	potato		2.6	0.0542	0.202	0.01	0.0125	Chaney et al., 1988
field		103	beetroot		0.4	0.0039	0.222	0.000862	0.0010777	Chumbley and Unwin 1982
field		97	leeks		0.8	0.0082	0.2	0.001649	0.0020619	Chumbley and Unwin 1982
field		176	potato		0.2	0.0011	0.222	0.000252	0.0003153	Chumbley and Unwin 1982
field		110	radish		2.9	0.0264	0.222	0.005853	0.0073159	Chumbley and Unwin 1982
field		107	salad onions		0.6	0.0056	0.125	0.000701	0.0008762	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	radish	0	92.4	0.0257	0.047	0.0012	0.0015	Cobb et al., 2000
smelter flue-dust	6.8	146.3	potato	0.2	0.2	0.0014	0.222	0.000303	0.0003794	Dudka et al. (1996)
smelter flue-dust	6.8	340	potato	0.2	0.4	0.0012	0.222	0.000261	0.0003265	Dudka et al. 1996
smelter flue-dust	6.8	2202.5	potato	0.2	0.7	0.0003	0.222	7.06E-05	8.82E-05	Dudka et al. 1996
smelter flue-dust	6.8	5452.5	potato	0.2	0.9	0.0002	0.222	3.66E-05	4.58E-05	Dudka et al. 1996
urban gardens-field			carrot				0.118	0.0006	0.00075	Finster et al., (2004)
urban gardens-field			onion				0.125	0.004	0.005	Finster et al., 2004
urban gardens-field			radish				0.047	0.00094	0.001175	Finster et al., 2004
indust. sewage-field-Egypt	ND	334	garlic		1	0.0030	0.387	0.001	0.00125	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	onion		1.1	0.0030	0.125	0.0004	0.0005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	radish		2.3	0.0070	0.047	0.0003	0.000375	Gorbunov et al., 2003
sewage sludge-field	70	259	carrot	0.33	0.48	0.0040	0.118	0.0005	0.000625	Hooda et al., 1997
Pb arsenate - grnhs	60.9	342.3	carrot	3.9	13.3	0.0400	0.118	0.005	0.00625	Hutchinson et al. (1974)
Pb arsenate - grnhs	60.9	342.3	onion	10	75.4	0.2000	0.125	0.03	0.0375	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	parsnip	7.8	14.8	0.0400	0.209	0.008	0.01	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	radish	7.9	27.5	0.0800	0.047	0.004	0.005	Hutchinson et al. 1974
field		14.49	carrot		0.92	0.0635	0.118	0.007492	0.0093651	Liu et al. 2006
field		14.49	leek		0.92	0.0635	0.146	0.00927	0.0115873	Liu et al. 2006
field		14.48	radish		0.47	0.0325	0.047	0.001526	0.0019069	Liu et al. 2006

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Env. contam. Soil (paint?) - potted - grnhs		2000	beet		19	0.0095	0.127	0.001	0.00125	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	carrot		34	0.0170	0.118	0.002	0.0025	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	turnip		22	0.0110	0.085	0.0009	0.001125	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	carrot		8.16	0.0490	0.118	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	radish		11.7	0.0710	0.047	0.0033	0.004125	Pandey and Pandey, 2009
smelter area - ag field	30	440	potato	0.099	0.099	0.0002	0.202	0.000045	5.625E-05	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	carrot	0.25	1.17	0.0013	0.118	0.00024	0.0003	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	leek	0.34	2.67	0.0031	0.146	0.00045	0.0005625	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	potato	0	0.15	0.0002	0.202	0.000034	0.0000425	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	radish	0	3.83	0.0044	0.047	0.00021	0.0002625	Pruvot et al., 2006
landfill-field		1000	carrot (unp)		5.1	0.0051	0.104	0.00053	0.0006625	Samsoe-Petersen et al., 2002
landfill-field		1000	potato (unp)		2	0.0020	0.113	0.00023	0.0002875	Samsoe-Petersen et al., 2002
landfill-field		1000	radish		7.4	0.0074	0.036	0.00027	0.0003375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	carrot (unp)		0.93	0.0070	0.118	0.0009	0.001125	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	potato (unp)		0.18	0.0010	0.222	0.0003	0.000375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	radish		1.65	0.0100	0.085	0.001	0.00125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		154	carrots			0.0030	0.118	0.0004	0.0005	Sauerbeck, 1991
sewage sludge-pot-field		154	radish			0.0200	0.05	0.0009	0.001125	Sauerbeck, 1991
field-wastewater	0.32	2.04	onion	0.22	0.46	0.2300	0.125	0.028	0.035	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	radish	0.28	0.73	0.3600	0.047	0.02	0.025	Shariatpanahi and Anderson 1986
flooded gardens		85.2	carrot		0.81	0.0095	0.118	0.001122	0.0014023	Sipter et al. 2008
flooded gardens		85.2	onion		1.06	0.0124	0.125	0.001555	0.001944	Sipter et al. 2008
non-flooded gardens		27.8	carrot		0.278	0.0100	0.118	0.00118	0.001475	Sipter et al. 2008
non-flooded gardens		27.8	onion		0.13	0.0047	0.125	0.000585	0.0007307	Sipter et al. 2008
smelter contam - field	22	163	potato		2.95	0.0180	0.202	0.0037	0.004625	Tomov & Alandjiyski, 2006
field - smelter	21.6	319.6	carrot			0.0320	0.108	0.003456	0.00432	Zheng et al. 2007a
field - smelter	21.6	319.6	turnip			0.0270	0.088	0.002376	0.00297	Zheng et al. 2007a
field - smelter		167	potato				0.11	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00403±0.0075

Table H.12-1 Mercury field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conver- sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Hgt pots -env. chamber		17.6	cabbage		1.5	0.09	0.08	0.007	0.00875	Caille (2005)
Hgt pots -env. chamber		17.6	rape		1.7	0.09	0.08	0.008	0.01	Caille et al., 2005
field-compost			lettuce				0.05	0.0122355	0.0152944	Cappon 1987
field-compost			spinach				0.08	0.0137064	0.017133	Cappon 1987
field-compost			Swiss chard				0.08	0.01201	0.0150125	Cappon 1987
field		4.77	amaranth		0.27	0.0566038	0.08	0.0045283	0.0056604	Liu et al. 2006
field		4.77	cabbage		0.21	0.0440252	0.08	0.003522	0.0044025	Liu et al. 2006
field		4.77	celery		0.31	0.0649895	0.08	0.0051992	0.006499	Liu et al. 2006
field		4.77	Ch cabbage		0.15	0.0314465	0.08	0.0025157	0.0031447	Liu et al. 2006
field		4.77	Ch chive		0.32	0.067086	0.08	0.0053669	0.0067086	Liu et al. 2006
field		5.5	leek		0.19	0.0345455	0.08	0.0027636	0.0034545	Liu et al. 2006
field		4.77	pakchoi		0.41	0.0859539	0.08	0.0068763	0.0085954	Liu et al. 2006
field-contam fungicide -greenhouse grown	ND	1.64	lettuce		0.173	0.10549	0.05	0.0052745	0.0065931	(MacLean, 1974)
field-contam fungicide -greenhouse grown	ND	7.13	lettuce		0.103	0.01445	0.05	0.0007225	0.0009031	MacLean 1974
sewage sludge - field		2.5	cabbage		0.01	0.004	0.08	0.0003	0.000375	Muntau et al., 1987
field-wastewater	0.06	0.16	basil	0.05	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	gard cress	0.04	0.12	0.75	0.08	0.06	0.075	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	mint	0.06	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	tarragon	0.04	0.13	0.81	0.08	0.065	0.08125	Shariatpanahi and Anderson 1986
flooded gardens		0.81	sorrel		0.06	0.0740741	0.08	0.0059259	0.0074074	Sipter et al. 2008
field - smelter	0.037	1.28	leek			0.139	0.08	0.01112	0.0139	Zheng et al. 2007a
field - smelter	0.037	0.76	Ch cabbage				0.055	0.016	0.02	Zheng et al. 2007a
field - smelter	0.037	1.5	Grn onion				0.085	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	1.5	spinach				0.088	0.005	0.00625	Zheng et al. 2007a
field - smelter	0.037	0.4	celery				0.058	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	0.5	cabbage				0.052	0.031	0.03875	Zheng et al. 2007a
field - smelter	0.037	0.5	lettuce				0.042	0.015	0.01875	Zheng et al. 2007a
field - smelter	0.037	0.3	mustard				0.071	0.01	0.0125	Zheng et al. 2007a

Average mercury uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0163±0.0202

Table H.12-2 Mercury field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			peppers			0.00222	0.126	0.0002797	0.0003497	Cambra et al. 1999
field-compost			broccoli				0.126	0.0145385	0.0181731	Cappon 1987
field-compost			cabbage				0.08	0.0120093	0.0150117	Cappon 1987
field-compost			cucumber				0.039	0.0002636	0.0003295	Cappon 1987
field-compost			pepper				0.074	0.0014145	0.0017681	Cappon 1987
field-compost			squash				0.082	0.0016629	0.0020787	Cappon 1987
field-compost			tomato				0.059	0.0036445	0.0045557	Cappon 1987
field		5.5	broccoli		0.12	0.0218182	0.126	0.0027491	0.0034364	Liu et al. 2006
field		4.03	cucumber		0.15	0.0372208	0.039	0.0014516	0.0018145	Liu et al. 2006
field		4.77	Eggplant		0.26	0.0545073	0.073	0.003979	0.0049738	Liu et al. 2006
field		4.77	kidney bean		0.27	0.0566038	0.111	0.006283	0.0078538	Liu et al. 2006
field		4.77	pepper		0.14	0.0293501	0.126	0.0036981	0.0046226	Liu et al. 2006
field		4.77	tomato		0.13	0.0272537	0.059	0.001608	0.00201	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	tomato	0.034	0.037	0.0071	0.059	0.00042	0.000525	MacLean 1974
field-wastewater	0.06	0.16	leek	0.04	0.1	0.63	0.12	0.075	0.09375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	squash		0.037	0.045679	0.082	0.0037457	0.0046821	Sipter et al. 2008
flooded gardens		0.81	tomato		0.01	0.0123457	0.059	0.0007284	0.0009105	Sipter et al. 2008
field - smelter	0.037	1.28	aubergine			0.003	0.066	0.000198	0.0002475	Zheng et al. 2007a
field - smelter	0.037	1.28	capsicum			0.007	0.081	0.000567	0.0007088	Zheng et al. 2007a
field - smelter	0.037	1.5	tomato				0.056	0.004	0.005	Zheng et al. 2007a
field - smelter	0.037	0.3	bitter melon				0.066	0.016	0.02	Zheng et al. 2007a

Average mercury uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00855±0.0194

Table H.12-3 Mercury field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conver- sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			broad beans			0.003506	0.126	0.0004418	0.0005522	Cambra et al. 1999
field-compost			bean				0.111	0.0011126	0.0013907	Cappon 1987
field	0.15	0.38	corn		0.011	0.0289474	0.261	0.0075553	0.0094441	Feng et al. (2006)
Hgt field-smelter-9 sites			brown rice			0.002	0.888	0.002	0.0025	Horvet et al., 2003
Hgt field-smelter-2 sites			brown rice			0.0001	0.888	0.00009	0.0001125	Horvet et al., 2003
Hgt field-clean area-2 sites			brown rice			0.009	0.888	0.008	0.01	Horvet et al., 2003
field		0.21	wheat		0.003	0.0142857	0.875	0.0125	0.015625	Huang et al. (2008)
HgCl ₂ - pots - chamber	ND		oats	0.009	0.013	0.002	0.917	0.0018	0.00225	John 1972
HgCl ₂ - pots - chamber	ND		peas	0.001	0.002	0.00033	0.257	0.000085	0.0001063	John 1972
Hgt field-smelter-23 sites		0.1782	corn		0.0061	0.03	0.261	0.0089	0.011125	Li et al., (2008)
pots - phenyl mercuric acetate	0.08	5.24	oats	0.113	0.163	0.031	0.917	0.029	0.03625	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	soybeans	0.074	0.076	0.015	0.925	0.013	0.01625	MacLean 1974
flooded gardens		0.81	bean		0.03	0.037037	0.111	0.0041111	0.0051389	Sipter et al. 2008
field - smelter	0.037	1.28	kidney bean			0.067	0.103	0.006901	0.0086263	Zheng et al. 2007a
field - smelter	0.037	1.5	cowpea				0.097	0.001	0.00125	Zheng et al. 2007a

Average mercury uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00804±0.0096

Table H.12-4 Mercury field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0104746	0.0130932	Cappon 1987
field-compost			carrot				0.118	0.0036308	0.0045385	Cappon 1987
field-compost			onion				0.125	0.0105478	0.0131847	Cappon 1987
field-compost			radish				0.222	0.0129371	0.0161713	Cappon 1987
field-compost			turnip				0.222	0.0056406	0.0070507	Cappon 1987
HgCl ₂ - pots - chamber	ND		carrot	0.044	0.053	0.0075	0.118	0.00089	0.0011125	John (1972)
HgCl ₂ - pots - chamber	ND		radish	0.013	0.026	0.02	0.085	0.0017	0.002125	John 1972
field		5.5	carrot		0.24	0.0436364	0.118	0.0051491	0.0064364	Liu et al. 2006
field		4.77	radish		0.21	0.0440252	0.2	0.008805	0.0110063	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	carrot	0.086	0.18	0.034	0.118	0.0041	0.005125	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	potato	0.047	0.055	0.01	0.222	0.0023	0.002875	MacLean 1974
field-wastewater	0.06	0.16	onion	0.06	0.06	0.38	0.125	0.047	0.05875	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	radish	0.04	0.08	0.5	0.085	0.043	0.05375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	carrot		0.02	0.0246914	0.118	0.0029136	0.003642	Sipter et al. 2008
flooded gardens		0.81	onion		0.02	0.0246914	0.125	0.0030864	0.003858	Sipter et al. 2008
field - smelter	0.037	1.28	carrot			0.044	0.108	0.004752	0.00594	Zheng et al. 2007a
field - smelter	0.037	1.28	turnip			0.034	0.088	0.002992	0.00374	Zheng et al. 2007a
field - smelter	0.037	0.3	potato				0.11	0.002	0.0025	Zheng et al. (2007b)

Average mercury uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0119±0.0167

Table H.13-1 Nickel field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (industrial sewage irrigation)		13.37	palak (spinach)		4.2	0.31	0.08	0.02	0.025	Kumar Sharma et al., (2007)
field (industrial sewage irrigation)		15.61	palak (spinach)		5.9	0.38	0.08	0.03	0.0375	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		14.52	palak (spinach)		2.6	0.18	0.08	0.02	0.025	Kumar Sharma et al., 2007
indust. Poll. Depo. - field		119.32	amaranthus		9.5	0.08	0.08	0.0064	0.008	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	spinach		10.62	0.089	0.08	0.0071	0.008875	Pandey and Pandey, 2009
landfill-field		49	lettuce		1.23	0.025	0.05	0.00125	0.0015625	Samsøe-Petersen et al., 2002
sewage sludge - field		120	cabbage		24	0.2	0.08	0.02	0.025	Muntau et al., 1987
sewage sludge-field	22.5	51.8	spinach	4.76	9.46	0.178	0.08	0.014	0.0175	Hooda et al., 1997
sewage sludge-field	28.1	34.6	spinach	0.88	1.2	0.03	0.08	0.003	0.00375	Jamali et al., 2007
sewage sludge-field			spinach				0.08	0.0048	0.006	Sridhara Chary et al., (2008)
urban gardens-field-to-greenhouse	10	50.7	lettuce	0.73	1.25	0.024	0.045	0.00108	0.00135	Sterrett et al., 1996

Average nickel uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0145 ± 0.0121

Table H.13-2 Nickel field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		112	peach		1.5	0.0133929	0.131	0.0017545	0.0021931	Basar and Aydmalp 2005
field		117	peach		1.6	0.0136752	0.131	0.0017915	0.0022393	Basar and Aydmalp 2005
field		122	peach		2	0.0163934	0.131	0.0021475	0.0026844	Basar and Aydmalp 2005
highly contam area		53	blackberry					0.0021	0.002625	Samsøe-Petersen et al., 2002
highly contam area		53	pear					0.0013	0.001625	Samsøe-Petersen et al., 2002
highly contam area		53	plum					0.0007	0.000875	Samsøe-Petersen et al., 2002

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
indust. Poll. Depo. - field		119.32	eggplant		7.92	0.066	0.073	0.0048	0.006	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	tomato		9.85	0.083	0.059	0.0049	0.006125	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	bell pepper		0.7	0.007	0.074	0.0005	0.000625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	cucumber		0.43	0.004	0.039	0.0002	0.00025	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	fig		1.6	0.02	0.225	0.0045	0.005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	olive		0.41	0.004	0.2	0.0008	0.001	Gorbunov et al., 2003
sewage sludge-field	28.1	34.6	Indian squash	1.3	2.1	0.06	0.082	0.005	0.00625	Jamali et al., 2007

Average nickel uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00293 ± 0.00226

Table H.13-3 Nickel field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (sewage-fed lake irrigation)			Beans			0.1	0.111	0.0111	0.013875	Lokeshwari and Chandrappa (2006)
highly contam area		53	hazelnut					0.033	0.04125	Samsoe-Petersen et al., 2002
indust. sewage-field-Egypt	ND	106	bean (spot)		6.9	0.07	0.894	0.06	0.075	Gorbunov et al., (2003)
indust. sewage-field-Egypt	ND	106	bean (white)		1.9	0.02	0.894	0.02	0.025	Gorbunov et al., 2003
landfill-field		49	green bean		6.37	0.13	0.076	0.0099	0.012375	Samsoe-Petersen et al., 2002
sewage sludge-field	28.1	34.6	cluster beans	1.21	2.1	0.06	0.111	0.007	0.00875	Jamali et al., 2007
sewage sludge-field	28.1	34.6	peas	1.12	1.18	0.03	0.257	0.009	0.01125	Jamali et al., 2007
sewage sludge-pot-field		25	beans			0.3	0.099	0.03	0.0375	Sauerbeck, 1991
sewage sludge-pot-field		25	peas			0.2	0.257	0.04	0.05	Sauerbeck, 1991

Average nickel uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0306 ± 0.0224

Table H.13-4 Nickel field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
indust. Poll. Depo. - field		119.32	carrot		3.65	0.031	0.118	0.0037	0.004625	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	radish		3.98	0.033	0.047	0.0016	0.002	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	garlic		2.6	0.02	0.125	0.003	0.00375	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	onion		3.1	0.03	0.125	0.004	0.005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	radish		3.8	0.04	0.085	0.003	0.00375	Gorbunov et al., 2003
landfill-field		49	carrot (unpeeled)		1.86	0.038	0.132	0.005	0.00625	Samsoe-Petersen et al., 2002
landfill-field		49	potato (unpeeled)		0.34	0.007	0.185	0.0013	0.001625	Samsoe-Petersen et al., 2002
landfill-field		49	radish		1.57	0.032	0.048	0.0015	0.001875	Samsoe-Petersen et al., 2002
sewage sludge-field	22.5	51.8	carrot	2.17	5.28	0.118	0.118	0.014	0.0175	Hooda et al., (1997)
sewage sludge-pot-field		25	carrots			0.08	0.118	0.009	0.01125	Sauerbeck, 1991
sewage sludge-pot-field		25	radish			0.2	0.05	0.01	0.0125	Sauerbeck, 1991

Average nickel uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00638 ± 0.00516

Table H.15-1 Selenium field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash	1.5	1.7	cabbage	0.07	0.2	0.1	0.08	0.009	0.01125	Furr et al. 1978
sewage sludge - field		0.4	cabbage		1.1	2.8	0.08	0.2	0.25	Muntau et al., 1987
field-compost			lettuce				0.05	0.008482	0.0106025	Cappon 1987
field-compost			lettuce				0.05	0.010372	0.012965	Cappon 1987

field		9.84	lettuce		19.16	1.94715	0.05	0.0973575	0.1216969	van Mantgem et al. (1996)
field		6.18	lettuce		5.61	0.90777	0.05	0.0453885	0.0567356	van Mantgem et al. 1996
field		15.9	lettuce		13.63	0.85723	0.05	0.0428615	0.0535769	van Mantgem et al. 1996
field		16.83	lettuce		27.9	1.65775	0.05	0.0828875	0.1036094	van Mantgem et al. 1996
field		17.37	lettuce		12.37	0.71215	0.05	0.0356075	0.0445094	van Mantgem et al. 1996
field-compost			spinach				0.08	0.016888	0.02111	Cappon 1987
field-compost			Swiss chard				0.08	0.00957	0.0119625	Cappon 1987

Average selenium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0587 ± 0.0713

Table H.15-2 Selenium field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to- wet wt conver- sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash-potted soil	0.3	1.2	apple (w/o seeds)	0.01	0.03	0.03	0.159	0.004	0.005	Furr et al. (1979)
field-compost			broccoli				0.126	0.0130125	0.0162656	Cappon 1987
field-fly ash-potted soil	0.3	1.2	cabbage	0.04	2.4	2	0.08	0.2	0.25	Furr et al. 1979
field-compost			cabbage				0.08	0.0216667	0.0270833	Cappon 1987
field-compost			cucumber				0.039	0.0010563	0.0013203	Cappon 1987
field-compost			pepper				0.074	0.0025107	0.0031384	Cappon (1987)
field-compost			squash				0.082	0.0027089	0.0033862	Cappon 1987
field-fly ash-potted soil	0.3	1.2	tomato	0.015	1.5	1.2	0.059	0.07	0.0875	Furr et al. 1979
field-compost			tomato				0.059	0.0099387	0.0124234	Cappon 1987
field-fly ash - pot	1.5	1.7	tomato	0.01	0.02	0.01	0.059	0.007	0.00875	Furr et al. 1978

Average selenium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0415 ± 0.0776

Table H.15-3 Selenium field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			bean				0.111	0.0070366	0.0087958	Cappon 1987
field-smelter		16.9	brown rice		1.06	0.06	0.888	0.056	0.07	Horvet et al., (2003)
field-fly ash - pot	1.5	1.7	bush bean	0.02	0.07	0.04	0.111	0.005	0.00625	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	bush bean	0.025	1.3	1.1	0.111	0.1	0.125	Furr et al. 1979
field-fly ash - pot	1.5	1.7	corn	0.02	0.05	0.03	0.895	0.03	0.0375	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	Japanese millet grain	0.025	1.4	1.1	0.888	1	1.25	Furr et al. 1979
field-fly ash-potted soil			onion		2.3	1.9	0.125	0.2375	0.296875	Furr et al. 1979

Average selenium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.256 ± 0.450

Table H.15-4 Selenium field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conversion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0098107	0.0122634	Cappon 1987
field-fly ash-potted soil	0.3	1.2	carrot	0.015	1.5	1.3	0.118	0.1	0.125	Furr et al. 1979
field-compost			carrot				0.118	0.0082179	0.0102723	Cappon 1987
field-fly ash - pot	1.5	1.7	carrot (peeled)	0.02	0.06	0.04	0.118	0.004	0.005	Furr et al. 1978
field-compost			onion				0.125	0.0550223	0.0687779	Cappon 1987
field-fly ash - pot	1.5	1.7	Onion (peeled)	0.02	0.21	0.1	0.125	0.02	0.025	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	potato	0.025	1.8	1.5	0.222	0.3	0.375	Furr et al. 1979
field-fly ash - pot	1.5	1.7	Potato (peeled)	0.02	0.03	0.02	0.222	0.004	0.005	Furr et al. (1978b)

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field-compost			radish				0.222	0.0391143	0.0488929	Cappon 1987
field-compost			turnip				0.222	0.0112321	0.0140402	Cappon 1987

Average selenium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0689 ± 0.114

H.11 Summary and Recommendations

OEHHA recommends the root uptake factors in Table H.16 for metals and metalloids.

Table H.16 Recommended Soil-to-plant uptake factors for inorganic metals and metalloids in edible crops^a

Element	Leafy	Exposed	Protected	Root
Arsenic	1×10^{-2}	2×10^{-2}	7×10^{-2}	8×10^{-3}
Beryllium	2×10^{-4}	8×10^{-3}	3×10^{-4}	5×10^{-3}
Cadmium	1×10^{-1}	2×10^{-2}	1×10^{-2}	8×10^{-2}
Chromium (VI)	3×10^{-1}	2×10^{-2}	7×10^{-2}	3×10^0
Fluoride	4×10^{-2}	4×10^{-3}	4×10^{-3}	9×10^{-3}
Lead	8×10^{-3}	7×10^{-3}	3×10^{-3}	4×10^{-3}
Mercury	2×10^{-2}	9×10^{-3}	1×10^{-2}	2×10^{-2}
Nickel	1×10^{-2}	3×10^{-3}	3×10^{-2}	6×10^{-3}
Selenium	6×10^{-2}	4×10^{-2}	3×10^{-1}	7×10^{-2}

^a Soil-to-plant UFs represent the fresh weight concentration of a contaminant in the plant part over the wet weight concentration of contaminant in the soil.

H.12 References

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Appendix I. Fish
Bioaccumulation Factors

I.1 Introduction

The algorithm used in the AB-2588 risk assessment to estimate exposure to contaminants via intake of angler-caught fish contains a chemical-specific variable known as a bioaccumulation factor (BAF). Fish are exposed to chemicals that are deposited into their aqueous environment from airborne sources. Only a small subset of Hot Spots chemicals are wholly or partially in the particulate phase and thus subject to deposition. These chemicals include semivolatile organic chemicals and toxic metals. Table I-1 presents the chemical-specific BAF values derived by OEHHA for the Hot Spots program. This appendix outlines the methods used for estimating BAFs and summarizes the available literature used for deriving the chemical-specific BAFs recommended in Table I-1.

Table I-1. Recommended Default Fish BAFs for Edible (Muscle) Tissue^a

Organic Chemicals^b	
Diethylhexylphthalate (DEHP)	40
Hexachlorobenzene (HCB)	80,000
Hexachlorocyclohexanes (HCH)	3000
Polycyclic aromatic hydrocarbons (PAH)	800
Polychlorinated biphenyls (PCB)	2,000,000
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	300,000
Inorganic and Organic Metals^c	
Arsenic	20
Beryllium	40
Cadmium	40
Chromium	20
Lead	20
Mercury	
Inorganic mercury	80
Methylmercury	6,000,000
Nickel	20
Selenium	1000

^a All BAFs were rounded to the nearest whole number.

^b Lipid-normalized to adult rainbow trout with 4% lipid content in muscle tissue, and based on the freely dissolved fraction of organic chemical in water under conditions of average POC and DOC in U.S. lakes and reservoirs.

^c Based on wet weight muscle tissue concentration, and on the total water concentration of the metal or metalloid in water, with the exception of methyl mercury, which assumes a translator of 3.2% for freely dissolved MeHg in water compared to the total Hg water concentration.

Accumulation of a chemical in fish is a physical-chemical process by which chemicals tend to apportion themselves between the fish and the fish's contact with its environment. The environment in this case is defined broadly to include the water, food that the fish eats, and contact with materials other than water. Accumulation of chemicals in fish may result in human exposure from fish consumption, which may be significant relative to other exposure pathways considered in the Hot Spots Program.

The Hot Spots program previously only considered the physical-chemical transfer of chemicals from the water column to the fish. This approach does not address other potentially important sources of toxic contaminant contributions to fish and can thus underestimate human exposure for some chemicals. This issue is discussed in more detail below.

The BAF reflects the uptake and retention of a chemical by fish from all surrounding media (e.g., water, food, sediment) when a steady-state concentration has been reached between the fish and the media. The BAF will vary depending on the organ or tissue of interest, but is also often expressed as the chemical accumulation in the whole fish. The BAF is defined under the Hot Spots program as representing the ratio of a concentration of a chemical in edible tissue, specifically the whole muscle tissue or muscle lipid fraction, to its concentration in the surrounding water in situations where the organism and its food are exposed and the ratio does not change substantially over time. The BAF is calculated as:

$$\text{BAF} = C_t / C_w \quad \text{Eq. I.1}$$

where:

C_t = concentration of the chemical in wet tissue

C_w = concentration of chemical in water

Lipophilic, organic chemicals tend to concentrate in the lipid fraction of fish and the resulting BAF is often lipid normalized to express the concentration of chemical in lipid (see below). The concentration of a chemical in water is often expressed in milligrams or micrograms of chemical per liter of water (i.e., mg/L or $\mu\text{g/L}$) and the concentration in tissue is often expressed in μg of chemical per kg tissue ($\mu\text{g/kg}$, or ppb). The BAF can be represented as a unitless factor through conversion of a volume of water to a mass (1 L water \approx 1 kg), or simply represented in L/kg.

In some instances, the BAF may be based on a bioconcentration factor (BCF). The BCF is defined as representing the ratio of a concentration of a chemical in tissue to only its concentration in the surrounding water when a steady-state concentration has been reached between the two media. Potential fish exposure via food sources is not included. Laboratory accumulation studies often determine BCFs due to the simplicity of the test and easier comparison with other BCF studies. Currently, U.S. EPA (2003a) recommends use of BCFs only for exposure to inorganic metals, presumably because intake of inorganic metals by fish via food sources is minor compared to uptake from water. However, a review of the literature by OEHHHA suggests contaminated food sources can also be an important source of metal accumulation in fish tissues. Thus, reliance on BCFs to estimate fish exposure may also underestimate the actual accumulation of a metal in fish.

For semi- or non-volatile organic chemicals that are highly persistent and hydrophobic (generally with a $\log K_{ow} > 4$), the magnitude of bioaccumulation by fish via food sources

can be substantially greater than the magnitude of bioaccumulation via exposure to water. For such chemicals, only true BAFs adequately assess accumulation of the chemical in fish tissues. For many of these persistent organic chemicals, biomagnification can occur. Biomagnification is the process through which chemical concentrations in fish increase as the chemical moves up the food chain, essentially through food sources. This process occurs because there are fewer organisms feeding off of more organisms at each level in the food chain, thus concentrating the chemical contaminants.

Numerous variables can affect uptake of persistent organic chemicals and inorganic metals in fish, therefore literature sources that reflected potential chemical accumulation as might occur under the “Hot Spots” program were our primary focus. That is, BCF/BAFs were primarily based on the edible portion (i.e., muscle tissue) of freshwater sport fish common to California lentic environments. Lentic environments consist mainly of standing water bodies including lakes, reservoirs and ponds. Sport fish that are caught and consumed in California are predominantly in trophic levels 3 and 4. These fish are typically of highest economic value and include predatory and carnivorous fish that feed on lower trophic level animals. BAF values for trophic level 2 organisms (e.g., zooplankton and larval fish stages) and non-sport fish, such as mosquito fish and the fathead minnow, were not considered unless there was a lack of accumulation data for higher trophic level sport fish.

The muscle tissue is defined here as the edible tissue of fish, although some ethnic groups may also eat various organs of fish. OEHHA’s California fish advisories recommend against eating the liver and other organs of fish, because they may have higher concentrations of organic contaminants than the muscle tissue (OEHHA, 2003). In addition, most inorganic metals will also concentrate in the organs, particularly the kidney and liver. Thus, the BAFs derived in this document cannot be used for estimating accumulation of chemicals in organs other than muscle tissue, as doing so could seriously underestimate the dose received by consuming fish organs and tissues other than muscle.

In California, common freshwater sport fish caught for consumption include various species of trout, catfish, bass, perch, sunfish and carp (CDFG, 2007). Mean muscle lipid content and trophic level data for some sport-fish are shown in Table I-2. In general, the size of the sport fish should be representative of the size being consumed by the target human population. Thus, the mean values are based on fish sizes that are caught and consumed by anglers. As Table I-2 shows, both muscle lipid content and trophic level can increase with increasing length (and age) of the fish. In some instances, lipid content or trophic level based on fish length, in cm, is provided.

Table I-2. Percent Muscle Lipid Content and/or Mean Trophic Level for some Freshwater Sport-Fish Found in California

Common Name	Mean % Muscle Lipid	Mean Trophic Level
<u>Carp (<i>Cyprinus carpio</i>)</u>	<u>4.45</u>	<u>3 (10-23 cm)</u> <u>2.4 (>23 cm)</u>
<u>Catfish</u>		
<u>Black bullhead</u>	<u>1.12</u>	<u>3</u>
<u>Brown bullhead</u>	<u>2.79</u>	<u>3</u>
<u>Channel catfish</u>	<u>5.00</u>	<u>3.1 (5-30 cm)</u> <u>2.8-4 (36-54 cm)</u>
<u>White catfish</u>	<u>2.15</u>	
<u>Yellow catfish</u>	<u>0.75</u>	
<u>Blue catfish</u>		<u>3</u>
<u>Flathead catfish</u>		<u>3.8</u>
<u>Perch</u>		
<u>Yellow perch</u>	<u>0.66</u>	<u>3.4</u>
<u>Trout</u>		
<u>Rainbow trout</u>	<u>4.00</u>	<u>3 (<30 cm)</u> <u>3.6 (30-50 cm)</u> <u>4 (>50 cm)</u>
<u>Brook trout</u>	<u>1.51</u>	<u>3.2</u>
<u>Brown trout</u>	<u>3.81</u>	
<u>Cutthroat trout</u>	<u>1.23</u>	<u>3 (<40 cm)</u> <u>3.2 (>40 cm)</u>
<u>Lake trout</u>	<u>10.90</u>	<u>3.7 (20-30 cm)</u> <u>3.9 (30-40 cm)</u> <u>4.2 (>40 cm)</u>
<u>Bass</u>		
<u>Smallmouth</u>	<u>1.1</u>	
<u>Largemouth</u>	<u>1.03 (35-48 cm)</u> <u>3.1 (54 cm)</u>	
<u>Black crappie</u>	<u>0.57 (14-23 cm)</u>	

Sources: U.S. EPA (1998); OEHA (1999); SFBWQCB (2005); Morrison et al. (1997)

1.1.1 Uptake and Accumulation of Semi- or Non-Volatile Organic Chemicals in Fish Tissues

Much of the field data for BAFs of organic chemicals comes from studies in the Great Lakes region (Eisenreich et al., 1981). The large surface area of the lakes, long hydraulic residence times, and major pollution sources near and upwind of the lakes have a significant impact on airborne deposited trace organic inputs.

For lipophilic, bioaccumulative organic chemicals, U.S. EPA (1998) recommends calculating a BAF based on the concentration of freely dissolved chemical in the ambient water and the lipid-normalized concentration in tissue. Regarding lipid

normalization, the BAF of lipophilic organic chemicals is usually directly proportional to the percent lipid content in the tissue of interest (U.S. EPA, 1998). For example, a fish with four percent lipid content would accumulate twice the amount of a chemical as a fish with two percent lipid content, all else being equal. Normalizing BAFs or BCFs to lipid content allows comparison between different fish species on the basis of factors other than percent lipid content. The lipid-normalized concentration is expressed as:

$$C = C_t / f \quad \text{Eq. I.2}$$

where:

C_t = Concentration of chemical in wet tissue (either whole fish or specified tissue)

f = Fraction lipid content in the organism

The lipid fraction of the edible muscle tissue is generally estimated because this is where the lipophilic chemicals will reside. However, the lipid content of muscle tissue can vary considerably among freshwater sport fish species (see Table I-1) as well as among the same species of different sizes and in different habitats. For this document, the rainbow trout lipid muscle content (4%) is used as the basis for point estimate BAFs for lipophilic organic chemicals. The rainbow trout is a common freshwater sport fish species caught and consumed in California and represents a reasonable “average” lipid content value among California sport fish. However, muscle lipid content can increase well above 10% in some fish species (carp, lake trout, and certain catfish) as they reach maximum size and age. The BAFs determined in this document may underestimate chemical intake if proportionally high consumption rates of such fish occur.

The tendency of an organic compound to bioconcentrate has been shown to be related to its lipophilicity and inversely related to the chemical’s water solubility. However, correlations between bioconcentration and physical properties are poor for very large molecules of high molecular weight and for chemicals metabolized by fish (Oliver and Niimi, 1985). Large molecules (about 300 to 500 MW) appear to be less efficiently transferred from water and food to fish tissues, but can have very long half lives in lentic/lotic environments (U.S. EPA, 2003a). Comparison of laboratory and field bioaccumulation studies in fish show that use of laboratory BCFs (kinetic and steady state studies) in which water was the only media for bioconcentration would severely underestimate the field residue levels of large organic molecules in fish, particularly if they are poor substrates for metabolic enzymes. This is a clear indication that water is not the primary route of fish exposure for these chemicals; consumption of contaminated food is likely the major chemical source.

U.S. EPA (1998) derived some BAFs from field measured biota-sediment accumulation factors (BSAFs) for very hydrophobic, organic compounds such as PCDD/Fs. The BSAF is the ratio of the lipid-normalized concentration of a chemical in tissue to its organic carbon-normalized concentration in surface sediment. Water concentrations of highly hydrophobic compounds can be difficult to measure accurately for field-measured BAFs, so U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of certain organic compounds in

ambient water. The California “Hot Spots” PCDD/F BAF point estimates discussed below in Section I.3.1.6 were derived from field-measured BSAF data by U.S. EPA (1998).

U.S. EPA (1998) recommends that for organic chemicals with a log K_{ow} greater than four, the concentrations of particulate organic carbon (POC) and dissolved organic carbon (DOC) in the ambient water should be either measured or reliably estimated. For these chemicals, the concentration of the chemical that is dissolved in ambient water excludes the portion sorbed onto particulate or dissolved organic carbon. The freely dissolved concentration is considered to represent the most bioavailable form of an organic chemical in water and, thus, is the form that best predicts bioaccumulation. The freely dissolved concentration is calculated as:

$$C^{fdw} = (f_{fd}) \times (C^{tw}) \quad \text{Eq. I.3}$$

Where:

C^{fdw} = Freely dissolved concentration of the organic chemical in ambient water

f_{fd} = Fraction of the total chemical in ambient water that is freely dissolved

C^{tw} = Total concentration of the organic chemical in ambient water

If F_{fd} is not known, it may be calculated using the equation:

$$F_{fd} = \frac{1}{1 + \text{POC} \times K_{ow} + \text{DOC} \times 0.08 \times K_{ow}} \quad \text{Eq. I.4}$$

For the California BAFs, DOC and POC were sometimes based on U.S. EPA (2003a) national default estimates of 2.9 mg/L for DOC and 0.5 mg/L for POC. These values reflect the central tendency estimated for DOC and POC for lakes and reservoirs distributed throughout the United States.

Field-based estimates of the freely dissolved concentration of an organic chemical in water (C^{fdw}) are preferred in order to predict BAF point estimates. However, Eq. I.4 was used to estimate f_{fd} in a number of instances when sufficient data was lacking in studies used to estimate a BAF.

I.1.2 Uptake and Accumulation of Inorganic and Organic Metals in Fish Tissues

In aquatic systems the availability of a metal to fish depends on many physico-chemical as well as biological factors. As summarized by Dallinger et al. (1987), availability is influenced by the chemical speciation of the ionic forms. The chemistry of the water including factors such as pH, hardness, and the presence of organic compounds and suspended particles may change the activity of free metal ions and influence the speciation of heavy metals. Binding to, and release from the sediment also affects the availability of metals to fish. Among the biological factors affecting metal availability, species-specific differences like feeding behavior and habitat preferences play a

dominant role. These basic features are modified by physiological factors, such as accumulation rates and the binding capacity in various fish species. The three ways by which inorganic metals may enter fish include body surface, the gills, and the alimentary tract. However, fish seem to be able to homostatically regulate some heavy metals that they are exposed to. Thus, BCFs and BAFs for metals will generally be smaller compared to BCFs and BAFs for persistent bioaccumulative organic chemicals.

In general, soluble metal fractions may accumulate preferentially via the gills, and particulate metal fractions via the alimentary tract (Dallinger et al., 1987). Unlike persistent, hydrophobic organic chemicals, bioconcentration and biotransference factors of metals tend to decrease with increasing trophic level up to fish, although the organometal methylmercury is an exception. However, even if biomagnification is not observed, or bioconcentration factors are small, the amount of metal transferred via food or water can be high enough to reach levels that are harmful to humans. This is because under chronic exposure of a water system, very high metal levels may occur in sediments, macrophytes and benthic animals in relation to the water levels. Thus, ingestion of sediment and sediment-dwelling invertebrates by bottom-dwelling fish species may be an important route of metal uptake by these fishes.

The wet weight muscle tissue concentrations of metals are used for determination of the BAF values. If the reference data is expressed only as a dry weight muscle tissue concentration, the tissue concentration was adjusted to a wet weight concentration using a factor of 0.24 (i.e., water content of fish muscle is roughly 75-76% by weight) if specific conversion data is not presented in the reference to calculate the adjustment.

An inverse relationship with metal accumulation and weight/size of the fish has been observed; metal in tissues decrease with increasing size or weight of fish (Liao et al., 2003). This affect has been attributed to growth dilution, increased metabolic rate in juvenile fish and increased ability to depurate the metals as the fish matures. As a result, metal uptake studies in fingerlings or juvenile fish may overestimate bioaccumulation of mature sport fish caught and consumed by anglers and were usually not used in this document to derive accumulation factors.

Another factor to take into account is exposure duration. Numerous accumulation studies summarized below have observed long exposure times, on the order of months, before steady-state levels of a metal is reached in fish tissues. Thus, short-term exposure studies may underestimate bioaccumulation of a metal in fish.

Based on the bioaccumulation literature for metals of interest in the "Hot Spots" program, some general statements can be made. Waterborne exposure to an inorganic metal will result in greatest metal accumulation in gill, kidney and liver. Metals in the diet will increase levels in the gut as well. Muscle tissue will have the lowest accumulation of the metals. Basing BAFs on whole body concentrations of a metal may overestimate metal intake, as the concentration of an inorganic metal can be quite high in the viscera (e.g., kidney and liver), with organ-specific BAFs of 1000 or greater. Where sufficient data was present, laboratory-measured BCFs were lower for a metal

than those derived using data from field studies. BCF studies often did not account for intake via contaminated food, which in some studies summarized below was shown to be an important route of exposure for inorganic metals. Also, many of the laboratory BCF studies likely did not attain steady-state concentrations because exposures were too short.

In almost all instances, acidic water bodies (generally with a pH of 6.5 or lower) will increase accumulation of the cationic metals and oxy-anionic chromium in fish organs and tissues compared to pH neutral (7.0 to 7.5) water bodies. The default BAFs in this document are primarily based on pH neutral lentic water bodies, as these are the most common in California. Consequently, the default BAFs may underestimate the actual accumulation of a metal in fish if the water body is acidic.

1.2 Derivation of Fish BAFs

1.2.1 Semi- or Non-Volatile Organic Chemicals

1.2.1.1 Diethylhexylphthalate (DEHP)

DEHP has been detected in marine and lake sediments, as well as in marine and freshwater sport fish (Stalling et al., 1973; McFall et al., 1985; Camanzo et al., 1987; Mackintosh et al., 2004). However, the source of the DEHP found in these marine and lake sediments is not likely to be solely from air emissions. The very high K_{ow} of 7.73 and model calculations suggests that DEHP could readily bioaccumulate in fish and that dietary uptake would be an important route of exposure (Staples et al., 1997; Gobas et al., 2003). However, bioaccumulation and biomagnification studies of DEHP in fish show roughly three orders of magnitude lower BCFs/BAFs than predicted based on the K_{ow} of DEHP. This finding is a result of trophic dilution and lack of biomagnification through the aquatic food web, primarily due to the metabolic transformation of DEHP in fish (Staples et al., 1997; Mackintosh et al., 2004). The term trophic dilution means that the BAF tends to decrease as the trophic level increases.

The only freshwater study from which a field-measured BAF was developed was based on a Dutch study investigating the occurrence of DEHP in the freshwater and fish throughout the Netherlands (Peijnenburg and Struijs, 2006). Twenty-five samples of bream and roach fish and 66 freshwater samples from 23 sites were collected throughout the country. Based on the geometric mean DEHP concentration of 1.8 $\mu\text{g/kg}$ wet fish and the dissolved freshwater DEHP concentration of 0.33 $\mu\text{g/L}$, a BAF of 5.5 is calculated (Table I.3). We corrected for the lipid fraction in the whole fish samples (median: 0.5% lipid), generating a lipid-normalized DEHP BAF of 1.1×10^3 . Finally, we also corrected for the muscle lipid content of rainbow trout (4%), which is approximately eight times greater than that of the bream and roach fish, generating a BAF of 44.

An assumption used for this BAF is that the influence of collecting fish and water samples at different times and from different locations on this BCF is not large. Another factor to consider is that the fish in the Dutch study was collected from both lentic and lotic water bodies. Lentic environments are characterized by still (not flowing) water, as in lakes and reservoirs. But the lotic environments are characterized by flowing water, as in streams and rivers.

Gobas et al. (2003) and Mackintosh et al. (2004) conducted a saltwater field study to assess the food-web bioaccumulation of a range of phthalate esters including DEHP. The calculated lipid-normalized BAF for the staghorn sculpin, a forage fish, and the dogfish, a predatory species, were 16,000 and 580, respectively (Table I.3). The larger dogfish (3 kg BW) has a smaller BAF than the sculpin (0.1 kg BW) due to gill elimination and fecal egestion rates dropping with increasing organism size and becoming negligible compared to growth rates.

Table I.3. BAF Values for DEHP in Fish

Fish Species	Total BAF^a	BAF(fd)^b	BAF(rt)^c
Staghorn Sculpin	ND ^d	16,000	640
Spiny Dogfish	ND	580	23
Bream & Roach	5.5	1091	44

^a Total concentration in whole fish divided by the total concentration of chemical in water

^b Freely dissolved, lipid-normalized concentration

^c BAF(rt) for sport-sized rainbow trout (rt) based on muscle lipid content of 4%

^d No data

Supporting studies from other laboratories report BCFs in small sport and non-sport fish. Whole-fish BCFs of 17 and 30 were estimated in separate studies in small rainbow trout (Mehrlle and Mayer, 1976; Tarr et al., 1990). Mayer (1976) estimates a BCF of 594 in fathead minnows, and Karara and Hayton (1984) estimated a BCF of 637 in sheepshead minnows. The estimated BCF values are based on the parent compound (i.e., they did not estimate a total BCF including DEHP and its metabolites) and did not include data that appeared to suffer from water solubility problems or lack of steady state attainment.

Basing the bioaccumulation of DEHP on BCF values does not take into account accumulation of DEHP from food or sediment sources, which may result in an underestimation of the BAF. In addition, basing a BAF on fingerlings or small fish may overestimate BAFs for sport-sized fish. Until field-based bioaccumulation studies for specific lentic water bodies are published for DEHP, we recommend that the BAF of 44, based on the Dutch freshwater field study, be used in the “Hot Spots” program as the default point estimate for DEHP accumulation in sport fish.

I.2.1.2 Hexachlorobenzene

HCB in the atmosphere is predicted to be predominantly in the vapor phase (see Appendix E). HCB concentrations in the vapor phase averaged 96.6% (range: 92-

100%) of the total HCB concentration in air samples over Ontario, Canada (Lane et al., 1992). This finding would suggest that airborne deposition of HCB into water bodies would be small enough to disregard. However, due to the extreme persistence of HCB in air, water and soil, accumulation of HCB into water bodies by both dry and wet deposition can be significant (Eisenreich et al., 1981; Kelly et al., 1991). Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from atmospheric sources, report HCB in water, sediment and fish tissue samples (Eisenreich et al., 1981).

Niimi and Oliver (1989) determined the percent lipid content and HCB concentration in muscle tissue of four salmonid species (brown, lake, and rainbow trout and coho salmon) collected from Lake Ontario. Based on the published water concentration of HCB in Lake Ontario, the researchers calculate a total BAF of 101,333. The total BAF was lipid-normalized based on 4% muscle lipid content in the fish, and adjusted for the concentration of freely dissolved HCB in water, assuming a DOC content of 0.25 mg/L in Lake Ontario from Gobas (1993). The resulting BAF(fd) is 2.6×10^6 .

We did not adjust the BAF(fd) to the muscle lipid fraction of rainbow trout (0.04) used in the California "Hot Spots" program because it is the same as the fish investigated by Niimi and Oliver (1989). We calculated the freely dissolved HCB fraction in water (0.78) from Eq. H.4 using the national default DOC and POC content of lakes and reservoirs (U.S. EPA, 2003a). A final BAF point estimate of 81,120 ($2.6 \times 10^6 \times 0.04 \times 0.78$) is recommended for California fish.

U.S. EPA (1998) calculates a similar BAF(fd) of log 6.40 (2.5×10^6) using Lake Ontario whole fish HCB data from Oliver and Niimi (1988). This BAF(fd) is similar to that estimated by Niimi and Oliver (1989) using only the muscle HCB concentration (BAF(fd) = 2.6×10^6) of the fish presented. U.S. EPA (1998) also calculated a mean log BAF(fd) of 5.70 (5.0×10^5) derived from BSAF data for HCB. Pereria et al. (1988) and Burkhard et al. (1997) determined a similar log BAF(fd) in the range of 6.03 to 6.68 for bioaccumulation of HCB in small, mostly non-sport fish in estuarine environments.

1.2.1.3 Hexachlorocyclohexanes

Technical grade hexachlorocyclohexane (HCH) generally consists of five isomers, including α -, β -, γ -, δ -, and ϵ -HCH. α -HCH is the most common isomer in technical grade HCH, and γ -HCH, also known as lindane, is most often isolated and used for its insecticidal action. Consequently, most environmental fate and bioaccumulation studies have investigated the α - and γ -isomers.

Lindane is a relatively small MW compound with a short half-life in fish, so rapid equilibrium occurs between the chemical concentration in fish and the water (Oliver and Niimi, 1985). The short half-life is probably a result of its log $K_{ow} < 4$. The high chlorine content of HCHs prevents metabolism of the isomers by rainbow trout (Konwick et al., 2006). The half-life of lindane in sport-sized fish (11-13 days) is longer than in juvenile fish (about 4 days). However, Geyer et al. (1997) report that α -HCH has a longer half-

life of 14.8 days in juvenile rainbow trout. In addition, they observed a positive correlation for fish lipid content and the BCF for lindane.

The major factor governing residue levels for HCHs appears to be the chemical concentration in the water (Oliver and Niimi, 1985). Thus, good agreement between field BAFs and laboratory BCFs in rainbow trout is achieved. For lindane, the whole-fish laboratory BCF was 1200 and the whole-fish field BAF in Lake Ontario fish was 1000. For α -HCH, the whole-fish laboratory BCF was 1600 and the whole-fish BAF in Lake Ontario fish was 700.

In a subsequent comprehensive investigation at Lake Ontario, Oliver and Niimi (1988) report total BAFs for α -HCH and lindane of 5357 and 9333, respectively. The lipid-normalized whole fish BAFs shown in Table I.4 were based on a weighted average lipid content of 11% for the four fish species examined (i.e., brown, lake, and rainbow trout, coho salmon).

Normalizing the BAFs to represent the freely dissolved fraction in water based on the national default DOC and POC values for lakes and reservoirs had little effect on the freely dissolved fraction of the HCHs, as chemicals with $\log K_{ow} < 4$ (the lindane and α -HCH $\log K_{ow}$ s are 3.67 and 3.78, respectively) will not partition significantly to OC. Normalizing the muscle concentration of the HCHs based on the muscle lipid content of rainbow trout (4%) results in point estimate BAFs of 3394 for lindane, and 1948 for α -HCH.

Table I.4. BAF Values Based on Lake Ontario Salmonids

HCH Isomer	Total BAF ^a	BAF(fd) ^b	BAF(rt) ^c
Lindane (γ -HCH)	9333	84,845	3394
α -HCH	5357	48,700	1948

^a Total concentration in whole fish divided by the total concentration of chemical in water

^b Freely dissolved, lipid-normalized concentration based on 11% lipid content in whole fish

^c BAF point estimates based on muscle lipid content of 4% for sport-sized rainbow trout

Niimi and Oliver (1989) determined the percent lipid content and HCH concentrations in muscle tissue, rather than only whole fish (apparently from the same fish examined in their previous study). The HCH concentrations in muscle adjusted for an average muscle lipid content of 4% for rainbow trout are 5.7 and 1.4 $\mu\text{g/kg}$ for α -HCH and lindane, respectively. Using the water concentrations of 2.8 and 0.3 ng/L for α -HCH and lindane, respectively, from Oliver and Niimi (1988) provides BAFs of 2036 (α -HCH) and 4667 (lindane).

Because the muscle HCH concentration data in Niimi and Oliver (1989) was at or below the limit of detection for some fish, particularly for lindane, the California BAF point estimate is based on the Oliver and Niimi (1988) data presented in Table I.4. We recommend a BAF(rt) point estimate of 2671 for the "Hot Spots" program, which is the arithmetic average of the muscle tissue BAF(rt)s for the two major HCH isomers in Table I.4.

1.2.1.4 Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are compounds with two or more fused benzene rings and often contain alkyl side groups. In water and sediment, low molecular weight PAHs (i.e., containing two or three aromatic rings) are more easily degraded by microbes, whereas the high molecular weight PAHs (i.e., containing four or more aromatic rings), including benzo[a]pyrene (BaP), tend to persist (Meador et al., 1995).

Bioaccumulation of PAHs in fish has not been rigorously studied, in part because PAHs undergo liver metabolism in fish resulting in low to non-detectable concentrations of the parent PAHs in fish tissues (Meador et al., 1995). Bioaccumulation of PAHs tends to decline with increasing K_{ow} , probably due to low gut assimilation efficiency and increased metabolism. However, low molecular weight PAHs tends to be less persistent in fish than the high molecular weight PAHs, probably due to more ready diffusion in and out of lipid pools.

BaP has been shown to be extensively metabolized in fish. In small bluegill sunfish (4 to 12 g wet weight) exposed to ^{14}C -labelled BaP in water, only 5% of the radiolabel in whole fish samples at the end of 24 hr exposure was found to be the parent compound (McCarthy and Jimenez, 1985). In their risk assessment, Boyce and Garry (2003) estimated a whole fish BCF of 14 for BaP based on the average value reported from relevant laboratory bioaccumulation studies in the literature.

Using the assumption that a typical lipid fraction of whole fish is 0.05 (Staples et al., 1997), and a muscle/whole body lipid ratio of 0.20 for adult rainbow trout (Niimi and Oliver, 1983), we calculated the lipid-normalized muscle tissue BCF as 56 for BaP. Adequate data for the DOC and POC water concentrations were not supplied by the studies used to derive the BCF, so the influence of this factor on the BAF could be accounted for in the final estimate.

Burkhard and Lukasewycz (2000) determined field-measured BAFs for several PAHs found in water, sediment and lake trout muscle lipid of Lake Superior. The total BAF and BAF(fd) in Table I.5 were calculated by the researchers for lake trout in Lake Superior. The BAF(rt) was calculated by OEHA for PAHs in rainbow trout (4% muscle lipid content) using default DOC + POC content for U.S. lakes and reservoirs. The relative order of metabolism was obtained by dividing the BAF of the chemical by its corresponding K_{ow} . By increasing rate of metabolism in the fish, the relative order was pyrene, benz[a]anthracene, chrysene/triphenylene, fluoranthrene, and phenanthrene. Thus, metabolism of the parent PAH compound appears to primarily control accumulation in the muscle tissue.

Table I.5. BAF Values for Polycyclic Aromatic Hydrocarbons

PAH congener (# of rings) ^a	PEF ^b	Total BAF ^c	BAF(fd) _d	BAF(rt) ^e
Phenanthrene (3)	ND ^f	18	89	4
Fluoranthrene (4)	ND	331	1660	62
Pyrene (4)	ND	10,471	52,481	2067
Benz[a]anthracene (4)	0.1	9550	53,703	1573
Chrysene/triphenylene (4)	0.01 (chrysene only)	759	4074	124 ^g

^a Number of benzene rings per PAH compound shown in parentheses

^b Potency Equivalency Factor for carcinogenicity, using benzo[a]pyrene as the index PAH compound with a PEF=1.

^c Total concentration in fillet of lake trout divided by the total concentration of chemical in water

^d Freely dissolved, lipid-normalized concentration based on 20.5% lipid content in fish fillet samples

^e BAF point estimates based on muscle lipid content of 4% for rainbow trout and default DOC + POC content for U.S. lakes and reservoirs from U.S. EPA (2003a).

^f Not determined, as a result of inadequate or no evidence for carcinogenicity in animals.

^g Assumed to represent BAF(rt) for both chrysene and triphenylene

The data in Table I.5 suggests that PAHs with four rings are more likely to accumulate in fish than PAHs with three rings. A study by Zabik et al. (1996) found some five- and six-ring PAHs in muscle fat of lake trout from Lake Superior. This study did not detect BaP in the fish tissue, but did find dibenzo[ah]pyrene which has a potency equivalency factor (PEF) value of 10. BAFs could not be calculated for any PAHs with five or more rings, either because dissolved levels of these congeners could not be detected in the water, or because the congener could not be detected in the fish (Baker and Eisenreich, 1989; 1990; Zabik et al., 1996). Another reason is that the individual PAHs quantified in water and fish were not all the same between various studies.

We calculated an average BAF(rt) of 849 from the congener groups in Table I.5 that have PEFs (i.e., benz[a]anthracene and chrysene), and is recommended as the default point estimate of BAF(rt) for PAHs. Considering that measurable levels of high molecular weight carcinogenic PAHs have been detected in fish muscle (although not enough data is present to estimate BAFs), but that a BAF for BaP is likely below the BAF(rt) of 849, a point estimate based on the most bioaccumulative carcinogenic PAHs should be sufficiently health protective to avoid underestimation of a BAF for the carcinogenic PAHs.

I.2.1.5 Polychlorinated biphenyls (PCBs)

PCBs are a group (209 congeners) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. However, probably less than 100 congeners are found at concentrations of significance in commercial PCB mixtures and environmental samples, and fewer represent a toxicological concern (Niimi, 1996). Solubilities and octanol-water partition coefficients (K_{ow}) for PCB congeners range over several orders of magnitude. The K_{ow} s, which are often used as

estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Since log K_{ow} values of most PCB congeners are higher than 5, biomagnifications through trophic transfer is the primary mechanism governing the accumulation of these compounds in fish (Oliver and Niimi, 1985; van der Oost et al., 2003). Thomann and Connolly (1984) demonstrated that more than 99% of PCBs in Lake Michigan lake trout came from food. A food web bioaccumulation PCB study by Morrison et al. (1997) noted that over 99% of PCB 153 accumulated in fish through consumption of contaminated food and 79.9% of PCB 42 accumulation was through food (PCB 42 has a lower K_{ow}).

Food-web relationships and biomagnification may be more related to the PCBs in sediment rather than water. Therefore, biota sediment accumulation factors (BSAF) have been developed for PCBs as an indicator of bioavailability to fish because sediment is an important source for hydrophobic chemicals such as PCBs (Niimi, 1996). However, the PCBs found in the highest concentrations in fish generally reflected their high concentrations in water and sediment (Oliver and Niimi, 1988).

In the comprehensive field study by Oliver and Niimi (1988), the most common classes of PCB isomers in various salmon and trout species from Lake Ontario were the penta- and hexachlorobiphenyls, making up about 65% of the total isomeric composition. The tetra- and heptachlorobiphenyls made up another 30% of the isomeric composition. Eleven single and co-eluting PCB congeners (153, 101, 84, 138, 110, 118, 180, 87 + 97, 149, 187 + 182, and 105) constituted over half the PCBs in fish. The single most common congener was 153 (2,2', 4,4',5,5'-hexachlorobiphenyl). The tri, tetra, and penta congeners comprised a much higher fraction in water than in the fish. Thus, the PCB accumulation pattern in fish is not an accurate reflection of the aqueous composition of the mixture found in the lake.

Because the calculated total BAFs for the most common PCBs accumulating in fish gave a roughly 10-fold range for the values, a weighted average total BAF was calculated for the four most common chlorinated classes of PCB congeners in fish from the study by Oliver and Niimi (1988). These were the tetra-, penta-, hexa-, and hepta-CBs, which constituted about 95% of the overall PCBs accumulated in whole fish. The resulting weighted-average total BAF was 6.12×10^6 .

We calculated a lipid-normalized BAF of 5.56×10^7 based on the whole fish lipid content of 11% determined in the study by Oliver and Niimi (1988). The mean percent contribution of PCB congeners was similar for whole fish and muscle among the species even though total concentrations vary widely (Niimi and Oliver, 1989). Consistency among congener contribution in whole fish and muscle was also demonstrated by cumulative percent of the more common PCB congeners. The freely dissolved PCB portion in water is based on data by Gobas (1993) who found about half of total PCBs in Lake Ontario water was in the freely dissolved form. The resulting calculated lipid-normalized, freely dissolved BAF, or BAF(fd), is 1.11×10^8 .

Next, we adjusted the BAF(fd) to generate a BAF point estimate to be used in the California “Hot Spots” program. Correcting the BAF(fd) for the muscle lipid fraction of 0.04 in rainbow trout, and correcting for the freely dissolved PCB fraction in water (0.25, or 50% of that calculated for Lake Ontario) gives a final BAF point estimate of 2.22×10^6 ($1.11 \times 10^8 \times 0.04 \times 0.50$).

I.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans (PCDDs and PCDFs)

PCDDs and PCDFs are two groups of toxic compounds composed of 135 and 75 individual isomers, respectively. Most studies have focused on the 17 congeners with lateral Cl substitutions at the 2,3,7,8 positions (Niimi, 1996). These congeners appear to be primarily responsible for the accumulation and toxicity of PCDD/Fs. The 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF congeners were common in four fish species (brown trout, lake trout, rainbow trout, coho salmon) examined from Lake Ontario. Dietary uptake of PCDD/Fs appears to be of more importance than waterborne uptake, although dietary absorption efficiencies in fish are consistently lower and more variable compared to PCBs.

The two main lateral substituted PCDDs, 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, constituted about 89% of the sum of all PCDDs in the fish (Niimi, 1996). The two main PCDFs, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF, constituted 51% of the sum of all PCDFs in the fish. Since these congeners are the most bioaccumulative and have the greatest toxicity concern, the PCDD/F BAFs will be representative of these four congeners.

U.S. EPA (1998) derived lipid-normalized, freely dissolved BAFs (i.e., BAF(fd)) from field measured BSAFs. The high hydrophobic nature of PCDD/Fs makes it difficult to accurately determine field-measured BAFs (i.e., based on water concentrations) for this group of chemicals. U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of these compounds in ambient water. Using a weighted-average approach for the main congeners found in fish, the BAF(fd)s were 1.00×10^7 and 5.50×10^6 for PCDDs and PCDFs, respectively.

We then adjusted the BAF(fd)s to generate BAF point estimates to reflect the muscle lipid fraction of rainbow trout (0.04) for the “Hot Spots” program. The final BAF point estimates of 400,000 and 220,000 were calculated for PCDDs and PCDFs, respectively, for California fish. The average BAF of these two values, 310,000, is the recommended BAF point estimate for the “Hot Spots” program.

I.2.2 Derivation of Fish BCFs – Metallic and Organometallic Compounds

I.2.2.1 Arsenic

Inorganic arsenic (As), either as As(III) or As(V), are the predominant forms in aquatic ecosystems such as sediment and water, but organoarsenic compounds may be present at significant levels in freshwater fish. Average concentrations of As in ambient

freshwater are generally <1 to 10 µg/L (U.S. EPA, 2003b). U.S. EPA (2003b) states that recent research shows each of the major inorganic and organic As species, including As(III), As(V), arsenobetaine (AsB), dimethylarsenic acid (DMA), and monomethylarsonic acid (MMA), may exhibit different toxicities, and it may be important to take into account the fraction of total As present in the inorganic and organic forms when estimating the potential risk posed through consumption of As-contaminated fish. Ideally, the most appropriate BAFs would incorporate the most bioavailable and toxic form(s). This is currently not possible, so the point estimate BAF in this document will be based on total As in sport fish muscle tissue.

Direct accumulation of As in tilapia was proportional to the concentration of arsenicals in water (Suhendrayatna et al., 2002). Approximately 25% of absorbed arsenic from water in whole fish as either As(III) or As(V) were transformed to methylated arsenic, primarily methyl-, dimethyl-, and trimethyl- forms. Whether absorbed as As(III) or As(V) from water, metabolism in fish resulted in roughly equivalent concentrations of both inorganic arsenic species in whole fish, although As(III) was absorbed more easily than As(V).

Accumulation and transformation of As in the food chain has been investigated. In a three-step freshwater food chain (algae-shrimp-tilapia), exposure to As(III) in water resulted in total As concentrations decreasing in the organisms with each step up the food chain (Suhendrayatna et al., 2002). Inorganic As species were the predominant forms in each organism (As(III), 9-41%; As(V), 50-90%), with only a limited degree of As methylation at each step in the food chain. However, when As(V) was the dominant As species in water, mouthbreeder fish raised long-term in aquaculture ponds contained predominantly organoarsenic species in muscle tissue, with inorganic As equaling only 7.4% of total As (Huang et al., 2003).

Predicted and measured As concentrations in major organs of tilapia from culture ponds high in As observed highest As concentrations in the alimentary canal, blood and liver, and lowest concentrations in muscle tissue (Liao et al., 2005). Steady-state concentration of As in muscle tissue took up to 300 days to be achieved.

Arsenic bioaccumulation studies in fish have been conducted in laboratory, aquaculture pond, and field investigations, although exposure durations to achieve steady-state concentrations in fish tissues were only observed for the aquaculture and field studies. The BAFs findings are presented in Table I.6.

In aquaculture studies, an average BCF of 8.2 (range: 5.4 to 11) was determined for bioconcentration of As in muscle of mouthbreeder fish raised long-term in ponds from three different regions in Taiwan (Huang et al., 2003). The fish were collected from ponds containing 14.4 to 75.8 µg/L As in water. A BCF of 3.5 was recorded for As in muscle tissue of large-scale mullet raised in a Taiwanese aquaculture pond (Lin et al., 2001). In farmed tilapia fish exposed to As in water for 300 days, a muscle BCF = 4 was calculated (Liao et al., 2005). In a similar study, BCFs of 15 and 53 were obtained for As from tilapia muscle raised in two aquaculture ponds containing 49.0 and 17.8

µg/L As in water, respectively (Liao et al., 2003). Because the fish in these aquaculture studies were fed with artificial bait that did not contain As, the accumulation factors may better represent BCF values rather than BAF values.

Only two field studies were located that presented data to determine a muscle tissue BAF for fish in As-contaminated lentic water bodies. A BAF of 28 was determined from muscle tissue of the common carp exposed to As in four wastewater treatment basins in Pennsylvania (Skinner, 1985). Channel catfish and large-mouth bass from a reservoir impacted by mining and agricultural runoff had muscle BAF values of 12.5 for As (Baker and King, 1994).

Table I.6. BAFs for Arsenic in Muscle Tissue of Fish from Lentic Water Bodies

Location	Species	Arsenic Water Concentration	Arsenic Muscle Concentration	BAF	Reference
Taiwanese Aquaculture Studies					
Putai Pond	mouthbreeder	75.8 µg/L	0.41 µg/g	5.4	Huang et. al., 2003
Yichu Pond	mouthbreeder	15.1	0.12	7.9	Huang et. al., 2003
Hsuehchia Pond	mouthbreeder	14.4	0.16	11.1	Huang et. al., 2003
Putai Pond	large-scale mullet	169.7	2.41	14.2	Lin et. al., 2001
Hsuehchia Pond	tilapia	17.8	0.95	53.4	Liao et. al., 2003
Yichu Pond	tilapia	49.0	0.75	15.3	Liao et. al., 2003
Tilapia farms	tilapia	94	1.5	16	Liao et al., 2005
Field Studies					
San Carlos Reservoir, AZ	large-mouth bass	8	0.1	12.5	Baker & King, 1994
San Carlos Reservoir, AZ	channel catfish	8	0.1	12.5	Baker & King, 1994
Wastewater treatment basins, PA	common carp	3.0 – 16.0	0.22 - <0.05	28	Skinner, 1985

Among the studies presented in Table I.6, average BCF/BAFs were calculated for six fish species: 8.1 for mouthbreeder, 14.2 for large-scale mullet, 28 for tilapia, 12.5 for large-mouth bass and channel catfish, and 28 for common carp. The arithmetic average BAF combined for all species is 17, which we recommend as the BAF point estimate for As.

1.2.2.2 Beryllium

Little information could be found for bioaccumulation of beryllium in fish. U.S. EPA (1980) estimated a BCF of 19 in whole bluegill after 28 days of exposure in water. It is unknown if steady state levels were attained in the fish, although the whole-body elimination half-life was observed to be one day. Limited data by Eisler (1974) suggests that whole-fish accumulation of inorganic beryllium in mummichogs from seawater is similar to some other cationic metals such as cadmium, in that whole fish uptake of beryllium appears to be a passive process.

No information could be found regarding the accumulation of beryllium in muscle tissue of fish. Based on BCF and BAF studies of other cationic metals discussed in this appendix, steady state levels were probably not reached in bluegills during the 28-day exposure (U.S. EPA, 1980). The muscle BAFs for other cationic metals (i.e., cadmium, inorganic mercury, lead, nickel) presented in Table H.2 range from 20 to 80. We recommend that a mean cationic metal BAF of 40 be used for beryllium in sport fish until more comprehensive bioaccumulation studies are conducted.

1.2.2.3 Cadmium

A considerable number of cadmium (Cd) bioaccumulation studies have been carried out in fish. Freshwater sport fish accumulate Cd mainly in gills, kidney, liver, and gastrointestinal tract (Sangalang and Freeman, 1979; Harrison and Klaverkamp, 1989; Spry and Wiener, 1991; Szebedinszky et al., 2001). However, Cd does not accumulate as appreciably in muscle tissue of exposed sport fish and the concentration is generally low relative to other tissues and organs.

The Cd concentration in fish varies with the proportion of free divalent Cd in water, typically increasing with increasing water concentration (Camusso et al., 1995). Direct uptake across the gills has been generally considered the primary influx of the metal for fish in dilute waters (Spry and Wiener, 1991). However, absorption of Cd from contaminated food sources can be a significant route of exposure, and may be the dominant source of Cd in bodies of water with high pH and calcium levels (Ferard et al., 1983; Harrison and Klaverkamp, 1989; Farag et al., 1994; Kraal et al., 1995; Thomann et al., 1997).

The main characteristics of lakes that enhance bioaccumulation of Cd in fish include low pH (pH ≤ 6), low aqueous calcium (often < 2 mg/L), and low DOC (usually < 3 mg/L) (Spry and Wiener, 1991). In the eastern U.S., whole-body Cd levels in bluegill fish from low pH lakes were as much as 10-fold higher compared to cadmium in bluegills from circumneutral-pH lakes. In addition, accumulation of Cd in fish is more sensitive to changes in water hardness, usually expressed in mg/L CaCO₃, rather than changes in DOC (Wiener and Giesy, 1979).

Steady-state equilibrium of Cd in muscle and other tissues was obtained in brook trout at about 20 weeks exposure in a three-generation exposure study by Benoit et al.

(1976). Benoit et al. (1976) also recorded a muscle BCF = 3.5 in brook trout exposed to aqueous Cd in Lake Superoir water for 70 weeks. Equilibrium of Cd in tissues was also reached at 20 weeks of exposure.

Perhaps significantly, the numerous laboratory studies that measured muscle Cd content show an inverse relationship with water hardness. In several laboratory studies, BCFs varied between 1.6 to 4.8 for Cd in muscle of rainbow trout, carp and brook trout with a water hardness between 33 and 93 mg /L CaCO₃ (Benoit et al., 1976; Giles, 1988; Harrison and Klaverkamp, 1989; de Conto Cinier et al., 1997). Exposure durations for these studies ranged from 3 to 17 months, and tissue and organ Cd concentrations increased with increasing exposure duration. Two other laboratory studies that recorded somewhat higher BCFs of 17-19 in muscle of rainbow and brook trout also had the lowest water hardness (19-22 mg /L CaCO₃) (Sangalang and Freeman, 1979; Kumada et al., 1980). The exposure duration of fish to Cd-contaminated water for both of these studies was about 3 months. Alternatively, laboratory studies exposing rainbow trout to Cd in water with considerably higher hardness (140-320 mg/L CaCO₃) at circumneutral-to-high pH (7.4-8.2) for up to 80 weeks recorded BCFs from 0 to 2 in muscle tissue (Roberts et al., 1979; Calamari et al., 1982; Brown et al., 1994; Szebedinszky et al., 2001).

The level of DOC in the water of the laboratory BCF studies above were not discussed, but were likely low. Low DOC levels would allow water hardness to be the main factor affecting bioaccumulation of Cd.

Although comparatively few field studies have been published that investigated Cd accumulation in muscle tissue of sport fish, the field study by Wiener and Giesy (1979) supports the assumption that water hardness (and perhaps pH) is a more important factor in controlling tissue accumulation than the DOC content. In this study, a Cd muscle BAF = 12 was determined in bluegill stocked in an acidic (pH = 4.6), highly organic pond for 511 days. Measured total organic carbon of the pond was anywhere from 15 to >30 mg/L, but the CaCO₃ content of the pond was very low, averaging 2.1 mg/L.

Two field studies examined the effect of acidified water in New York lakes on fish tissue levels of various heavy metals as a result of acid deposition (i.e., acid rain) (Heit et al., 1989; Stripp et al., 1990). In general, higher BAFs were recorded for Cd in muscle tissue of yellow perch and white sucker from the most acidic lentic water body, Darts Lake, compared to two other lakes, Rondaxe and Moss lakes, with higher pH values (Table I.7). All three lakes were clear-water lakes with comparable concentrations of DOC.

Table I.7. BAFs for Cadmium in Muscle Tissue of Fish from U.S. Lakes

Location	Species	Lake pH	Cd Water Concentration (µg/L)	Cd Muscle Concentration (µg/g)	BAF
Darts Lake (1)	White sucker	4.9-5.4	0.7	0.062	89
Darts Lake (1)	Yellow perch	4.9-5.4	0.7	0.048	69
Darts Lake (2)	White sucker	5.1-5.4	0.26	0.038	146
Darts Lake (2)	Yellow perch	5.1-5.4	0.26	0.028	108
Rondaxe Lake (1)	White sucker	5.8-6.7	1.1	0.024	22
Rondaxe Lake (1)	Yellow perch	5.8-6.7	1.1	0.024	22
Rondaxe Lake (2)	White sucker	5.8-6.7	0.61	0.025	41
Rondaxe Lake (2)	Yellow perch	5.8-6.7	0.61	0.038	62
Moss Lake (1)	White sucker	6.5-6.8	0.6	0.022	36
Moss Lake (1)	Yellow perch	6.5-6.8	0.6	0.034	56
Skinface Pond, SC (3)	Bluegill	4.6	0.17	0.0021	12

Sources: (1) Stripp et al., (1990); (2) Heit et al., (1989); (3) Wiener and Giesy (1979).

The few field studies examining muscle tissue levels of Cd in contaminated lakes indicate that basing a BAF on laboratory BCF studies would underestimate the accumulation potential of Cd in fish. However, it is probably not appropriate basing a BAF on data from highly acidified lakes (i.e., Darts Lake and Skinface pond), as California generally does not have the lake acidification problem that exists in the northeastern U.S. Thus, we recommend default BAF point estimate for Cd of 40 based on fish from the variable pH (Rondaxe Lake) and circumneutral lakes (Moss Lake), which is the arithmetic average BAF combining both fish species (white sucker and yellow perch, which represent trophic level 3 and 4 fish, respectively) from these lakes.

I.2.2.4 Chromium

Hexavalent chromium (Cr(VI)) in water readily penetrates the gill membrane of fish and is the main route of uptake (Holdway, 1988). Organs and tissues that accumulate Cr(VI) include gills, spleen, kidney, gall bladder, gastrointestinal tract, opercular bone, and brain. Accumulation in muscle tissue is minor compared to these other tissues. No biomagnifications occur at higher trophic levels. Cr(VI) uptake is a passive process with resulting tissue concentrations directly proportional to exposure concentrations. Chromium bioavailability to fish increases with decreasing pH (7.8 to 6.5), resulting in increased bioaccumulation in tissues and organs (Van der Putte et al., 1981).

In a laboratory study, six-month exposure of rainbow trout to Cr(VI) as potassium dichromate ($K_2Cr_2O_7$) in water resulted in a muscle tissue BCF of 3 (Calamari et al., 1982).

A small freshwater aquatic ecosystem containing adult catfish was created in a small tank, and a single dose of potassium dichromate was added to the system (Ramoliya et al., 2007). After 21 days of exposure, a muscle tissue BCF <1 was calculated for the

catfish based on the average water concentration of Cr(VI) over the 21 days. However, the Cr(VI) content in the catfish had not reached equilibrium at the end of exposure, and was still increasing with increasing exposure duration. High levels of Cr(VI) in the intestine of the catfish suggests Cr(VI) may be absorbed via food sources.

Rainbow trout that were reared for two years in either a hatchery or river water that was contaminated with low levels of sodium dichromate had muscle tissue BCFs of 40 and 12, respectively (Buhler et al., 1977). Exposing the same fish to high concentrations of Cr(VI) (2.5 mg/L) for 22 days increased muscle levels of Cr(VI), but the resulting BCF was only 0.1-0.2.

Two field studies from South Africa determined the bioaccumulation of chromium in muscle tissue of fish. In adult African sharptooth catfish, muscle tissue BAFs of 10 and 16 were calculated for fish kept in a treated sewage maturation pond and in a reservoir, respectively, for 12 months (Van den Heever and Frey, 1996). Nussey et al. (2000) calculated an average muscle tissue BAF of 23.6 in the moggel, a cyprinid fish, collected from a different reservoir over a period of 15 months.

Based on the long-term field exposure studies, an average muscle BAF of 26 was calculated for rainbow trout in the Buhler et al. study, and an average muscle BAF of 13 was calculated for the African sharptooth catfish in the van den Heever and Frey study. Combined with the muscle tissue BAF of 23.6 in the moggel from Nussey et al. (2000), we calculate an arithmetic mean BAF of 21 and recommend this value as the BAF point estimate for Cr.

1.2.2.5 Lead

Similar to Cd, factors that may increase accumulation of cationic metals such as lead in fish include low pH (6.0-6.5 or less) in the water body, low concentrations of aqueous calcium that compete with lead for absorption through the gills, and low DOC (Varanasi and Gmur, 1978; Spry and Wiener, 1991; Lithner et al., 1995). Pb appears to have a greater tendency than Cd to associate with DOC and particulate matter in lake water, with accumulation in fish varying inversely with the concentration of dissolved organics in water (Wiener and Giesy, 1979). When Merlini and Pozzi (1977a) added a Pb salt to lake water, only 8% remained in the ionic form with the remainder presumably associating with dissolved organics.

Accumulation of Pb by fish typically increases with increasing exposure concentration in water, although Pb does not biomagnify in aquatic food chains (Spry and Wiener, 1991). Pb chiefly accumulates in the bone, scales, gill, kidney, and liver. Pb does not accumulate as appreciably in skeletal muscle tissue of fish. Primary mode of absorption has been suggested to be direct uptake of Pb in the ionic state across the gills, with lead from food sources being minor or insignificant (Merlini and Pozzi, 1977a; Spry and Wiener, 1991; Farag et al., 1994). On the other hand, another laboratory study found that lead uptake in fish via food was significant, if not more important than uptake via water (Vighi, 1981).

In a three-generation laboratory study, a BCF of 2 to 3 was estimated for Pb in muscle tissue of first and second generation brook trout (Holcombe et al., 1976). Exposure to Pb in water was for 38 and 70 weeks in first and second generation fish, respectively. The concentration of Pb in muscle had reached equilibrium at about 20 weeks of exposure.

Whole bluegill Pb concentrations has been shown to be as much as 10 times higher in bluegills from low-pH lakes ($\text{pH} \leq 6.0$) compared to bluegills from circumneutral-pH lakes ($\text{pH} 6.7-7.5$) (Spry and Wiener, 1991). In another study, whole-fish Pb levels in sunfish increased almost three-fold when lake water pH was decreased from 7.5 to 6.0 (Merlini and Pozzi, 1977b).

In other field studies, Pb accumulated to greater extent in muscle of white suckers and yellow perch from an acidic lake compared to more neutral lakes (Heit et al., 1989; Stripp et al., 1990) (Table I.8). With increasing lake acidity, muscle bioaccumulation of Pb became increasingly higher in bottom-dwelling, omnivorous white suckers compared to carnivorous yellow perch. Thus, contact with sediments by bottom-dwelling fish increases Pb bioaccumulation.

A considerably greater concentration of Pb was found in surface sediments (880-1005 $\mu\text{g/g}$) of the lakes compared to the water (2.0-3.0 ng/g) (Stripp et al., 1990). It was postulated that higher levels in fish tissues from acidic lakes result from increased mobilization of the cationic Pb species from sediments coupled with an increase in the cationic Pb species in the acidic water.

Table I.8. BAFs for Lead in Muscle Tissue of Fish from Lentic Ecosystems

Location	Species	Lake pH	Pb Water Concentration	Pb Muscle Concentration	BAF
Acidic water bodies					
Darts Lake (1)	White sucker	4.9-5.4	3.0 µg/L	0.13 µg/g	43
Darts Lake (1)	Yellow perch	4.9-5.4	3.0 µg/L	0.058	19
Darts Lake (2)	White sucker	4.9-5.4	1.5	0.13	87
Darts Lake (2)	Yellow perch	4.9-5.4	1.5	0.055	37
Acidic lakes & ponds, NJ (3)	Yellow perch	3.7-4.6	0.8 – 3.6	0.067 – 0.11	40
Variable and circumneutral water bodies					
Rondaxe Lake (1)	White sucker	5.8-6.7	2.0	0.048	24
Rondaxe Lake (1)	Yellow perch	5.8-6.7	2.0	0.058	29
Rondaxe Lake (2)	White sucker	5.8-6.7	2.3	0.050	22
Rondaxe Lake (2)	Yellow perch	5.8-6.7	2.3	0.050	22
Moss Lake (1)	White sucker	6.5-6.8	2.5	0.031	12
Moss Lake (1)	Yellow perch	6.5-6.8	2.5	0.024	10
Witbank Dam, South Africa (4)	Moggel	ND*	140	2.00	14

Sources: (1) Stripp et al. (1990), (2) Heit et al. (1989), (3) Sprenger et. al. (1988), (4) Nussey et al. (2000)

* No data

The field data indicate higher muscle BAFs in fish from highly acidified lakes (Table I.8). California generally does not have the acidification problem that exists in the northeastern U.S. Thus, a BAF point estimate for Pb was based on fish from the variable pH and circumneutral lakes. The BAF data from Nussey et al. (2000) was also included, although water pH data was not provided in the report. We calculate an arithmetic average BAF of 19 combining all fish species (white sucker, yellow perch and moggel) from these lakes and recommend this value as the Pb BAF point estimate.

I.2.2.6 Mercury (inorganic) and Methylmercury

Mercury, like other metals deposited into water, can occur in a number of physical and chemical forms. Physically, mercury can be freely dissolved or bound to organic matter or particles suspended in water. Mercury can be found as elemental mercury (Hg^0), inorganic ionic mercury (primarily Hg^{++}), or organic mercury (e.g., methylmercury (MeHg) or dimethylmercury).

Mercury (Hg) enters aquatic ecosystems primarily as inorganic Hg, but MeHg is the dominant form of Hg found in muscle tissue of freshwater fish (Spry and Wiener, 1991). MeHg has been shown to constitute virtually all, about 99% or greater, of the total Hg in

muscle of trophic level 3-4 freshwater sport fish even though much of the Hg analyzed in the water was in inorganic Hg (Bloom, 1992; Kuwabara et al., 2007). In whole fish, the proportion of inorganic Hg is greater (5% or more of total Hg) because whole body samples include visceral tissue, such as kidney and liver, which is the principal site of inorganic Hg accumulation in fish (Hill et al., 1996; Watras et al., 1998).

As summarized by Southworth et al. (2004), MeHg is produced in aquatic environments by the action of microorganisms on inorganic Hg. It can also be removed from the aquatic systems by microorganisms that demethylate MeHg. Once formed, MeHg is taken up by microorganisms, primary producers, aquatic invertebrates, and fish. MeHg in the organisms show the classical biomagnification process, with MeHg concentration increasing with trophic level. The concentrations of MeHg that are accumulated in fish are greatly affected by the nature of the aquatic food chain, and are sensitive to factors such as aquatic community composition and productivity. In many waters, minute concentrations (<10 ng/L) of waterborne inorganic Hg are capable of sustaining MeHg production at rates high enough to support bioaccumulation of MeHg in fish to levels warranting fish consumption advisories. The concentration of MeHg and inorganic Hg are positively related in natural waters, which would appear to support expressing a BAF for MeHg in fish as a ratio based on total or dissolved inorganic Hg in water. Calculating MeHg bioaccumulation in fish using such a ratio would be ideal for the “Hot Spots” program (i.e., estimate the concentration of dissolved MeHg in water based in the total Hg concentration deposited in water), but introduces another level of uncertainty compared to development of BAFs directly from published reports.

Using the dissolved MeHg fraction in water to derive BAFs is recommended, as this is the primary form of MeHg that is bioaccumulated in fish. MeHg is also more toxic than other forms of mercury. However, dissolved MeHg was not always the form measured in the studies U.S. EPA (2001) identified for inclusion in their database. Thus, translators were necessary to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. For lentic systems (i.e., lakes, reservoirs and ponds), the translators that may be use in the Hot Spots program include dissolved MeHg (MeHg_d) over the total Hg (Hg_t) and the MeHg_d over the total MeHg (MeHg_t). The lentic U.S. EPA translators are $\text{MeHg}_d / \text{Hg}_t = 0.032$, $\text{MeHg}_d / \text{MeHg}_t = 0.61$.

U.S. EPA (2001) derived the mean dissolved MeHg/total Hg translator of 3.2% for lentic ecosystems, and used it to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. Thus it can be interpreted that 3.2% of inorganic Hg that has deposited into a lake will be converted by microorganisms and found in the form of dissolved MeHg.

Table I.9 presents various BAFs for methylmercury from U.S. EPA (2001) and California data (OEHHA, 2006). Although U.S. EPA presents the geometric means of BAFs, OEHHA recommends the use of arithmetic means of the BAFs to provide a more health protective estimate. In developing their BAFs, U.S. EPA assumed that 100 percent of the mercury measured as total mercury in both trophic levels 3 and 4 was MeHg. This assumption provides a more health protective estimate.

Table I.9. Methylmercury BAFs for Lentic/Lotic^a Ecosystems from U.S. EPA and California Data

Agency	Environment/Comments	Mean	Trophic Level	
			3	4
U.S. EPA	Lentic Only	Geometric	1.1×10^6	5.7×10^6
U.S. EPA	Lentic Only	Arithmetic	1.5×10^6	6.2×10^6
California	Lentic Alternative	Geometric	NP	NP
California	Lentic Alternative	Arithmetic	NP	NP
U.S. EPA	Lotic Only	Geometric	5.7×10^5	1.2×10^6
U.S. EPA	Lotic Only	Arithmetic	1.3×10^6	3.9×10^6
California	Lotic Alternative	Geometric	6.8×10^5	1.1×10^6
California	Lotic Alternative	Arithmetic	1.4×10^6	3.5×10^6
U.S. EPA	Lentic/Lotic Combined	Arithmetic	1.4×10^6	5.0×10^6

^a Lentic environments are characterized by still (not flowing) water, as in lakes and reservoirs. Lotic environments are characterized by flowing water, as in streams and rivers.

In California, using a MeHg BAF developed by U.S. EPA is complicated by the large number of Hg point sources originating from legacy mining activities, a situation somewhat unique to California. Atmospheric deposition of Hg into water bodies may be overshadowed by the existing Hg already present due to legacy mining. In addition, very little published data exists for California lentic ecosystems in order to determine if total Hg concentrations are good predictors of MeHg concentration. The BAFs and translators developed by U.S. EPA were based primarily on atmospheric deposition of Hg into water bodies. Hg speciation in water and fish may be quite different depending on whether the Hg originated from mining or atmospheric deposition.

Nevertheless, OEHHA (2006) found that the national values predicted California fish MeHg concentrations very well except for some water bodies where Hg concentrations in water were statistically higher. Hg concentrations (≥ 0.2 ng/L) in these water bodies were found to be more than one standard derivation from the mean for other data used in these tests. We concluded that the national default values for BAFs and translators may not work well for all water bodies in California. However, based on the limited comparisons possible, BAFs and translators based on the California data and international studies (U.S. EPA database) were found to be similar.

In partial support, Kelly et al. (1995) observed that total Hg concentration was not a good predictor of MeHg concentration in stream water or in lakes in general, but it appeared to be a good predictor for lakes within individual geographic areas. In lotic ecosystems, Southworth et al. (2004) concluded that it is not valid to assume that the fraction of total waterborne Hg comprised by MeHg would remain constant while total Hg varies at high total Hg concentrations (roughly >50 ng/L) typical of systems affected by point-source or legacy contamination. However, at total Hg concentrations less than 10 ng/L, the %MeHg varies little. They postulated that such a relationship results from saturation of the ecosystems capacity to methylate inorganic Hg at high total Hg concentrations.

Although OEHHA does not currently have an oral chronic Reference Exposure Level for methyl mercury, OEHHA recommends using a translator of 3.2% to convert total Hg deposited in water to dissolved MeHg in water under the “Hot Spots” program. Additionally, the MeHg BAF = 6,200,000 (log 6.79) from Table I.9 is recommended for California sport fish caught and consumed from lentic ecosystems.

Inorganic Hg is absorbed by fish less efficiently than MeHg from both food and water, but if absorbed, is eliminated more rapidly. For example, rainbow trout fed inorganic Hg-contaminated prey resulted in Hg predominantly accumulating in the intestines, and the Hg was not significantly absorbed into the body (Boudou and Ribeyre, 1985). During the decontamination phase, Hg that had accumulated in the intestines was rapidly excreted.

In water, the most important route for uptake of inorganic Hg in fish is likely the gills, with accumulation of Hg mainly in the gills, kidney and liver (Allen et al., 1988; Gottofrey and Tjalve, 1991). Whole-body accumulation of inorganic Hg in rainbow trout and carp increases with decreasing water pH from 9 to 5, but did not reach equilibrium during a 17-day exposure in water (Wakabayashi et al., 1987).

MeHg is the primary concern for estimating Hg bioaccumulation. Since relatively little of the Hg in fish muscle is in the inorganic form, there is very little field data to estimate a BAF for inorganic Hg.

In a laboratory tank study investigating the relationship between inorganic Hg body burden levels and toxicity, a mean muscle BCF of 84 was calculated in rainbow trout exposed to HgCl in water for 60 to 130 days (Niimi and Kissoon, 1994). Steady-state levels in muscle tissue were reached by 60 days of exposure to high levels of HgCl (64 µg/L); these levels were eventually lethal to the fish. Since most lakes of concern contain inorganic Hg levels in the ng/L to low µg/L range, such high exposure conditions may not reflect an ideal situation for estimating an inorganic Hg BAF. In addition, it has been found that food sources containing inorganic Hg are also important for fish Hg bioaccumulation (Hill et al., 1996).

U.S. EPA (2001) has used a national criteria of 51 ng/L of total Hg in water as a measure that may result in the MeHg concentration of concern of 0.3 µg/g in fish. Using the assumption that, at most, 1% of the MeHg concentration in fish muscle is actually inorganic Hg, a BAF of 59 for inorganic Hg is calculated ($0.3 \mu\text{g/g} (0.01) \div 51 \text{ ng/L}$). Although this BAF derivation is a rather crude estimate of the inorganic Hg BAF, the value is near that calculated from the BCF study (BCF = 84) by Niimi and Kissoon (1994). OEHHA recommends using the inorganic Hg BAF point estimate = 84 (rounded to 8×10^1) derived from the Niimi and Kissoon study.

1.2.2.7 Nickel

In aquarium tank studies, brown trout exposed to water containing radioactive nickel (^{63}Ni) showed the greatest accumulation of the metal in the gills, kidneys and liver, with relatively low accumulation in muscle tissue (Tjalve et al., 1988). The Ni concentration in muscle was related to the water concentration of Ni (Van Hoof and Nauwelaers, 1984). Similar to other cationic metals, increasing the acidity of water increases accumulation of Ni in fish.

A muscle BCF of 1.5 was recorded in the brown trout following 3 week exposure to Ni in a water tank. However, equilibrium of Ni between water and fish tissues had not been attained. Rainbow trout exposed to Ni in hard water (hardness = 320 mg CaCO_3/L) for six months accumulated little or no Ni in muscle tissue (BCF = 0.8-1.1) (Calamari et al., 1982).

In a field study, Nussey et al. (2000) calculated an average muscle tissue BAF of 19 in the moggel, a cyprinid fish, collected from a reservoir containing various heavy metals, including Ni, over a period of 15 months. Average muscle BAFs of 4 and 39 were calculated in common carp collected from two different wastewater treatment basins in Pennsylvania (Skinner, 1985). The acidity of the treatment basin water was not discussed, so it is unknown if water acidity played a role for the variation in BAF values.

In laboratory studies, accumulation of Ni in fish muscle tissues is relatively low compared to other inorganic metals discussed in this document. There are also relatively few published reports investigating fish bioaccumulation of Ni. Based on the BAFs from the two field studies by Nussey et al. (2000) and Skinner (1985), we calculated an arithmetic mean average BAF of 21 and recommend this value as a point estimate BAF for Ni.

1.2.2.8 Selenium

Selenium (Se) occurs in the environment in several oxidation states with different physicochemical and biological properties (Besser et al., 1993). Se from both natural and anthropogenic sources enters surface waters primarily as the highly soluble Se(IV) and Se(VI) oxidation states, which form selenite, SeO_3^{2-} , and selenate, SeO_4^{2-} , respectively. Organic selenides, Se(-II), including Se-amino acids and Se-proteins, methyl selenides, and other Se-substituted analogs of organosulfur compounds, are produced by biological reduction of selenite and usually occur at lower concentrations in water than inorganic Se species. Little information is available for organic selenides, so the BAF is based on total Se.

Se is an essential micronutrient for most aquatic organisms but is also toxic at relatively low environmental concentrations. It is reported that Se concentrations in fish muscle rarely exceed 1 ppm (wet weight) in the absence of exposure to Se from geologic sources or from industrial wastes (Cumbie and Van Horn, 1979).

Four-month exposure of juvenile bluegill and largemouth bass to selenite (Na_2SeO_3) in water resulted in BCF values of 288 and 153, respectively, and was independent of water temperature and hardness (Lemly, 1982). Accumulation of Se in muscle was relatively slow, reaching a steady-state concentration after 90 days of exposure in both fish species. Accumulation of Se in fish skeletal muscle was presumed to be a result of the high affinity of Se for sulfhydryl groups found on many organic molecules in muscle tissue. However, bioconcentration in muscle was quite low compared to BCF values for other organs and tissues. Lemly (1982) observed higher bioconcentration of Se in the spleen, heart, liver, kidney, gill, and erythrocytes.

In a food-chain study (algae-daphnids-bluegill), whole bluegill fry accumulated greater Se concentrations from food than from water in selenite-based exposures, and aqueous and food-chain Se bioaccumulation were approximately additive (Besser et al., 1993). However, in both aqueous and food-chain exposures based on selenite and selenate, Se bioaccumulation was greatest in algae and least in bluegills. Se concentrations in whole bluegill fry did not differ significantly between selenite and selenate treatments in either aqueous or food-chain exposures. Inorganic Se BCF values ranged from 13 to 106 in whole blue gill fry with 30- to 40-day exposures, although a steady-state concentration was not attained.

In a field study, Cumbie and van Horn (1979) analyzed muscle Se levels in various species of fish, primarily bluegill, other sunfish, carp and bullhead, during spring and summer from a reservoir with a high Se concentration. The range of muscle BAFs among all fish was 632 to 5450 with an arithmetic average of about 1780. Further research at the same reservoir observed muscle BAFs in warmwater sportfish (primarily various species of perch, catfish, sunfish and crappie) ranging from 739 to 2019 with an arithmetic average of 1351 (Lemly, 1985). There was evidence of biomagnification of Se through the food-chain, although when considering only muscle tissue of fish, levels of Se appeared to be similar to that of molluscs, insects, annelids and crustaceans found at the reservoir.

Lower Se BAFs of 124 and 216 were calculated in muscle of white suckers and yellow perch, respectively, from an acidic lake in New York (Stripp et al., 1990). Based upon geochemistry, Se would be expected to be less soluble in acidic lakes. BAFs of 454 and 490 were determined for Se in muscle tissue of crappie and carp, respectively, collected from a wastewater treatment basin in Pennsylvania (Skinner, 1985).

The accumulation data indicates Se uptake from both food and water results in accumulation of Se in muscle tissue, and that BAF/BCF values can be quite variable even between different fish species within the same water body. The two related field studies investigating Se accumulation in fish from a North Carolina reservoir (Cumbie and Van Horn, 1979; Lemly, 1985) gave an average BAF of 1566 ($1351 + 1780 / 2$) combining all trophic level 3 and 4 fish. Not including the data from the acidic lake, we calculate an arithmetic mean BAF of 1019 when the average BAF from the North Carolina reservoir is combined with the average fish BAF from the Pennsylvania wastewater treatment basin from Skinner (1985). In support, the BAF is within the

predicted intervals (at water Se concentrations above 0.5 µg/L) of the Se whole fish bioaccumulation model for lentic systems developed by Brix et al. (2005). We recommend a default point estimate BAF of 1000 for selenium for use in the Hot Spots program.

1.3 Non-Bioaccumulated Chemicals

Some organic “Hot Spot” chemicals in which a significant airborne fraction can be found in the particle phase do not appear to be bioaccumulated in fish. For example, although data show that methylenedianiline (MDA) exists partly in the particle phase and is persistent in soils, the low log Kow of 1.59 (HSDB, 2008) and rapid metabolism in higher trophic level animals (ATSDR, 1998) indicates this chemical will likely not bioaccumulate in fish tissues. In addition, unpublished evidence summarized in ATSDR (1998) suggests that MDA does not bioaccumulate in carp. Until published evidence shows otherwise, a fish BAF for MDA will not be included in the fish pathway in the “Hot Spots” program.

In addition, OEHHA is proposing that fluoride should not be included in the fish pathway because fresh weight fluoride concentrations in muscle or the fillet portion of fish were found to be less than the water concentration, regardless of the weight of the fish (Gikunju, 1992; Mwaniki and Gikunju, 1995).

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Appendix J

Lactational Transfer

J.1 INTRODUCTION

Some toxic chemicals in the environment can accumulate in a woman's body and transfer to her milk during lactation. Chronic exposure to pollutants that accumulate in the mother's body can transfer a daily dose to the infant much greater than the mother's daily intake from the environment. For example, the mother's milk pathway can be responsible for about 25% of total lifetime exposure to dioxins and furans (USEPA, 2000).

Several reviews have listed numerous toxic chemical contaminants in human breast milk (Abadin et al., 1997; Liem et al., 2000; van Leeuwen and Malisch, 2002; LaKind et al., 2005; Li et al., 2009). Many of these chemical contaminants are carcinogens and/or have non-cancer health impacts on people who inhale or ingest them. Data suggest that breast-feeding infants during the first two years of life have greater sensitivity to toxic chemicals compared to older children and adults (OEHHA, 2009).

Multiple chemical contaminants have been measured in breast milk or have properties that increase their likelihood of partitioning to milk during lactation. OEHHA grouped these chemicals into the following four major categories:

- 1) Persistent highly-lipophilic, poorly metabolized organic contaminants, such as polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs), and polychlorinated dibenzo-p-dioxins (PCDDs), are by far the most documented group. These, by virtue of their lipophilicity, are found almost entirely in the milk fat. PCBs, methyl sulfones, and hexachlorobenzene (HCB) methyl sulfones have also been measured in the lipid phase of breast milk.
- 2) Lipophilic but more effectively metabolized organic contaminants such as polycyclic aromatic hydrocarbons (PAHs) occur in breast milk. The PAHs are a family of over 100 different chemicals formed during incomplete combustion of biomass (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Some of the more common parent compounds have been measured in breast milk and research suggests that chronic exposure to PAHs produces stores in maternal fat that can transfer (carryover) to breast milk (Fürst et al., 1993; Costera et al., 2009).
- 3) Inorganic compounds, metals, and some organo-metallics, including the heavy metals arsenic, lead, cadmium, and mercury, have been found in breast milk. These inorganics are generally found in the aqueous phase and most are bound to proteins, small polypeptides, and free amino acids. The lipid phase may also contain some organometallics (e.g. methyl mercury) and metalloids (such as arsenic and selenium).
- 4) Chemicals with relatively low octanol:water partition coefficients such as phenol, benzene, halobenzenes, halophenols, some aldehydes and the more polar metabolites of PCBs, PAHs, and pesticides may occur in both the aqueous and lipid phases of breast milk.

Since this document supports risk assessments conducted under the Air Toxics Hot Spots program, we are primarily discussing Hot Spots chemicals emitted from stationary sources.

Many of these persistent chemicals are ubiquitous in the environment and are global pollutants found in low concentrations in air, water and soil. Because some of these chemicals bio-concentrate in animal fat, the primary pathway of exposure to breastfeeding mothers would be consumption of animal products such as meat, milk, and eggs. Nearby polluting facilities can be a local source of exposure and can add to the mother's body burden of contaminants from global pollution through multiple pathways.

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHA derived human lactation transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral routes of exposure from global pathways (e.g. air, cigarette smoke or diet) in the same or a similar human population.

Briefly, human milk transfer coefficients (Tco_{hm}) represent the transfer relationship between the chemical concentration found in milk and the mother's chronic daily dose (i.e. concentration ($\mu\text{g/kg-milk}$)/dose ($\mu\text{g/kg/day}$) under steady state conditions. In its simplest form, the biotransfer factor is:

$$Tco_{hm} = C_m / D_t \quad (\text{Eq. J-1})$$

where:

Tco_{hm} = transfer coefficient from ingested and inhaled media (day/kg)
 C_m = concentration of chemical in mother's milk ($\mu\text{g/kg-milk}$)
 D_t = total maternal dose through all exposure routes ($\mu\text{g/day}$)

Equation J-2 estimates the concentration of contaminants in mother's milk by incorporating the Tco in the following way:

$$C_m = [D_{inh} + D_{wi} + D_{food} + D_{si}] \times Tco_{hm} \quad (\text{Eq. J-2})$$

where:

D_{inh} = dose through inhalation ($\mu\text{g/day}$)
 D_{wi} = dose through drinking water ingestion ($\mu\text{g/day}$)
 D_{food} = dose through ingestion of food sources ($\mu\text{g/day}$)
 D_{si} = dose through incidental ingestion of soil ($\mu\text{g/day}$)

However, if separate biotransfer information is available for the oral and inhalation route, equation J-3 incorporates route-specific Tcos in the following way:

$$C_m = [(D_{inh} \times T_{co_{m_{inh}}}) + (D_{ing} \times T_{co_{m_{ing}}})] \quad \text{Eq. J-3}$$

where:

D_{ing} = dose through ingestion ($\mu\text{g}/\text{day}$)

$T_{co_{m_{inh}}}$ = biotransfer coefficient from inhalation to mother's milk

$T_{co_{m_{ing}}}$ = biotransfer coefficient from ingestion to mother's milk

These coefficients, applied to the mother's chronic daily dose estimated by the Hot Spots exposure model, estimate a chemical concentration in her milk (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.1-1 lists the transfer coefficients for dioxins, furans, dioxin-like PCBs, PAHs and lead that OEHHA has estimated from data found in the peer-reviewed literature and reviewed in this appendix.

Estimates of toxicant biotransfer to breast milk are ideally chemical-specific. Data necessary to develop a transfer model are available in the open literature for a limited number of chemicals. Therefore, for some toxicants OEHHA has modeled the transfer of a class of chemicals with similar physical-chemical properties using a single Tco when data in the open literature are lacking.

The Hot Spots exposure model can estimate long-term total dose from an individual facility or group of facilities through many pathways of contamination and routes of exposure to the mother and ultimately to her infant. In this appendix, "multipathway toxicants" refers to airborne-released chemicals that can cause exposure through pathways in addition to inhalation. The indirect exposure pathways evaluated under the Hot Spots program include incidental ingestion of contaminated soil, ingestion of contaminated home-raised meat and milk, surface

drinking water, homegrown produce, angler-caught fish and skin contact with contaminated soil.

Relative to the lifetime average daily dose to the infant from other exposure pathways in the Hot Spots exposure model, the dose of some chemicals from mother's milk will be negligible. However, the mother's milk pathway may be a substantial contributor to the estimated total lifetime cancer risk for some chemicals emitted from a Hot Spots facility. Exposure from global sources is expected to make up most (almost all) of a mother's toxicant body burden. Therefore, the contribution to a mother's toxicant body burden from a single Hot Spot facility is expected to be very small. Regardless of the mother's toxicant body burden from both local and global sources, the benefits of breastfeeding outweigh the risks to the infant exposed to these toxicants during breastfeeding. Breast-feeding has a number of universally accepted benefits for the infant as well as for the mother (Mukerjee, 1998).

We established transfer coefficients (Tcos) for individual congeners of PCDDs/Fs and dioxin-like PCBs, individual and summary carcinogenic PAHs and lead through equations J.1-1 through J.1-3. We used data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination rates remain constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate and do not change over the lactation period. Unless a study reported the geometric mean or median, we converted arithmetic mean and standard deviation to geometric mean and GSD.

In the following sections, we describe the methods for deriving specific Tcos from measurements of human milk intake and transfer estimates from studies of populations published in the open literature. In some cases, OEHHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHHA describes the method of adjustment in the text and table containing adjusted values.

J.2 Mothers' Milk Transfer Coefficients for PCDD/Fs and PCBs

Polychlorinated dibenzo-p-dioxins and furans (PCDD/Fs) are two series of almost planar tricyclic aromatic compounds with over 200 congeners, which form as impurities in the manufacture of other chemicals such as pentachlorophenol and PCBs. PCDD/Fs also form during combustion (e.g. waste incineration) and the breakdown of biomass (e.g. in sewage sludge and garden compost) (Liem et al., 2000). IARC has classified many dioxins and dioxin-like compounds as known or possible carcinogens (WHO, 1997; OEHHHA, 2009). Their carcinogenic potency is related to the potency of 2,3,7,8-TCDD in a toxic equivalent (TEQ) weighting scheme (OEHHHA, 2009).

The main exposure to PCDD/Fs in the general population from global sources is through the intake of food of animal origin. PCB exposure has been linked to fish consumption. For example, Jensen (1987) observed that congener distribution patterns in contaminated fish and human milk were very similar suggesting that one of the primary sources of human exposure to PCBs in the study population was ingestion of contaminated fish (Jensen, 1987).

Estimates of PCDD/F and PCB TEQ-intake from dietary sources contaminated by global sources can vary by 3 to 4-fold within some populations and by as much as 29-fold between populations (Liem et al., 2000; Focant et al., 2002). Exposure from diet can be at least an order of magnitude higher than intake from ambient air or cigarette smoking (i.e., 0.1 to 4 pg/day) (Liem et al., 2000).

J.2.1 Biotransfer of PCDD/Fs and PCBs to Human Milk

The potential health impacts from exposure to PCBs, PCDDs and PCDFs include carcinogenicity, developmental, endocrine disruption, reproductive toxicity, and neurotoxicity. These persistent, lipophilic compounds can accumulate in the fat of women, transfer to breast milk, and thus result in infant exposure. Some countries implemented measures to reduce dioxin emissions in the late 1980s (Liem et al., 2000). PCBs were banned in the late 1970's and are no longer used in commercial products. Nevertheless, following the PCB ban and efforts to reduce PCDDs, PCDFs emissions, these toxicants are still detected worldwide in human milk, although at declining levels.

The World Health Organization (WHO) has carried out a series of international studies on levels of approximately 29 dioxins and dioxin-like contaminants in breast milk. The first WHO-coordinated study took place in 1987-1988, the second round in 1992-1993 and the third round was initiated in 2000-2003. In the second round, in which concentrations of PCBs, PCDDs and PCDFs were determined in milk samples collected in 47 areas from 19 different countries, mean levels in industrialized countries ranged from 10-35 pg I-TEQ/g-milk (Liem et al., 2000).

Much lower levels (40% lower than 1993) were detected in the 3rd round (Liem et al., 1995; Liem et al., 2000; van Leeuwen and Malisch, 2002) WHO exposure study. Nevertheless, several recent investigators have continued to measure levels of dioxin-like compounds in breast milk (LaKind et al., 2004; Barr et al., 2005; Wang and Needham, 2007; Li et al., 2009). PCBs still appear in human milk and are still much higher than the total concentrations of PCDDs and PCDFs. Several studies report pg/g-fat levels of PCDD/Fs compared to ng/g-fat levels of PCBs (100 to 1000 times higher) measured in human milk (Chao et al., 2003; Chao et al., 2004; Hedley et al., 2006; Sasamoto et al., 2006; Harden et al., 2007; Wittsiepe et al., 2007; Raab et al., 2008; Todaka et al., 2008).

Thus, nursing infants have the potential to ingest substantial doses during the breastfeeding period, relative to typical total lifetime dose of these compounds from global sources. Consequently, this pathway of exposure may supply a substantial fraction of PCDDs and PCDFs (about 25%) of the infant's total lifetime dose of these compounds (USEPA, 2000). Several studies have detected higher levels of PCBs in the sera (Schantz et al., 1994), adipose tissues (Niessen et al., 1984; Teufel et al., 1990) and bone marrow (Scheele et al., 1995) of mostly breast-fed children relative to partially breast fed infants. These studies were conducted many years after PCBs were banned and no longer used in commercial products. Some investigators have reported a 4-fold greater level of PCBs in the blood of fully breast-fed compared to partially breast-fed infants (Niessen et al., 1984).

In another study, Abraham et al (1994, 1996, 1998) measured elevated PCB concentrations in nursing infants after approximately one year of feeding (Abraham et al., 1994; Abraham et al., 1996; Abraham et al., 1998). These authors reported levels of 34 to 45 ppt (pg TEQ/g blood lipid) among breastfed infants versus 3 to 3.3 ppt blood lipid PCDD/F TEQ concentrations among formula fed infants.

Numerous studies have measured dioxins, furans and dioxin-like PCBs in mother's milk (Liem et al., 2000). The twenty nine dioxin-like PCBs listed in Table J.2-1 are recognized by OEHHHA as carcinogens and have potency factors associated with them (OEHHHA, 2008). Concentrations of TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), the most toxic PCDD, are low relative to other PCDDs and more than 50% of the total PCDD content consists of Octa-CDD. Early studies found around 70% of the total Hexa-CDDs (HxCDDs) is 1,2,3,6,7,8-HxCDD, and the remainder is mainly 1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD (USEPA, 1998). These proportions have not shifted in recent studies (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

PeCDD (1,2,3,7,8 Penta-CDD) is always found in the emissions from waste incinerators (USEPA, 1998). Early studies indicated that the presence of 1,2,3,7,8-PeCDD with other PCDDs/PCDFs in human milk suggested that the major source of exposure came from waste incinerator emissions (Buser and Rappe, 1984; Rappe et al., 1985; Mukerjee and Cleverly, 1987). Note that these congeners are measurable in human milk currently (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

Levels of PCDFs in human milk tend to be lower than PCDDs. However, PCDFs dominate in particulates emitted by combustion sources, including hazardous waste incinerators, and are present in higher concentrations in the atmosphere than PCDDs (USEPA, 1998). HxCDDs/HxCDFs and HpCDDs/HpCDFs are prevalent in pentachlorophenol. Incineration of wood and other products impregnated with pentachlorophenol results in the formation of these congeners and emissions of hexa- and hepta-CDDs/CDFs. Both 1,2,3,7,8 and 2,3,4,7,8-

PeCDFs have been detected in human milk, but 90% of the PeCDFs is generally 2,3,4,7,8-PeCDF. 1,2,3,4,7,8- , and 1,2,3,6,7,8- HxCDFs. 2,3,4,6,7,8-HxCDFs. 1,2,3,4,6,7,8-HpCDF are also prevalent.

Several investigators have observed that dose, degree of chlorination, degree of lipophilicity, and molecular weight influence how much PCDD/F congener is absorbed through the lungs or gut, metabolized and transferred from blood to milk (Yakushiji, 1988; Abraham et al., 1998; Schechter et al., 1998; Kostyniak et al., 1999; Oberg et al., 2002; Wittsiepe et al., 2007).

Numerous studies have attempted to correlate exposure to individual dioxins, furans and dioxin-like PCBs from ingestion of contaminated food with levels in human biological samples such as blood and milk. Transfer from intake sources to human milk has often been estimated in the context of accidental or occupational exposures or after a substantial decline in environmental concentrations (Liem et al., 1995; Pinsky and Lorber, 1998; Liem et al., 2000; Focant et al., 2002; Furst, 2006; Milbrath et al., 2009). Steady state conditions are not reached in these studies because the half-lives of these compounds are in years and exposure changed considerably over the period evaluated in each study.

Others have attempted to model the relationship between maternal intake and concentration in mother's milk using an indicator compound such as TCDD (Smith, 1987; Lorber and Phillips, 2002). Less understood is the relationship between modeled and measured transfer estimates of individual dioxins, furans and dioxin-like PCBs. The following sections describe the sources of data and methods for deriving estimates of transfer for an array of dioxins, furans and dioxin-like PCBs that have accounted to some extent for the non-steady state condition and other confounders.

J.2.2 Oral Biotransfer

OEHHA located a series of studies conducted on the Dutch population that allows for an oral biotransfer estimate of dioxins and furans, and accounts for changing exposure conditions. In 1988, Albers et al collected and analyzed three hundred nineteen breast milk samples from women enrolled through 28 maternity centers located throughout the Netherlands. Maternity centers were selected based on geographic distribution and degree of urbanization. Human milk samples were analyzed for 17 PCDD/F congeners and 8 PCB congeners (Albers et al., 1996).

Liem et al (1995) took a similar approach to collect about 100 samples from first-time mothers enrolled in 1993 through maternity centers dispersed throughout The Netherlands. Based on information obtained from a questionnaire about characteristics of the study subject, investigators determined that the 1993 cohort appeared to be comparable to the cohort studied in 1988. With one exception,

(1,2,3,4,7,8- HxCDD), a consistent downward trend can be seen among congeners of PCDD/Fs and PCB-118 that were analyzed during both sampling periods, (Table J.2-1).

Table J.2-1: Summary Estimates of Dioxin-like Compounds Dietary Intake during Three Periods Over 15 years, and Human Milk Levels over Five Years in the Dutch Population

Chemical/ group	TEF	1978 (diet) ^a	1984/5 (diet) ^a	1994 (diet) ^a	1988 (milk) ^b	1993 (milk) ^a
		Mean, SD	Mean, SD	Mean, SD	Mean, SD	Mean, SD
		pg/d*	pg/d*	pg/d*	pg/kg- milk	pg/kg -milk
2,3,7,8- TCDD	1	13.2, 1.32	6, 2.94	3.6, 1.26	264,14	124, 56
1,2,3,7,8- PeCDD	1	39.6, 6.73	15, 4.65	4.8, 2.26	435,185	324, 116
1,2,3,4,7,8- HxCDD	0.1	85.8, 23.17	23.4, 17.55	7.2, 5.98	328,51	344, 192
1,2,3,6,7,8- HxCDD	0.1	325.8, 45.61	89.4, 42.02	19.8, 22.77	2445,349	1484, 668
1,2,3,7,8,9- HxCDD	0.1	105, 21.0	32.4, 21.38	10.8, 9.61	395,32	276, 132
1,2,3,4,6,7, 8-HpCDD	0.01	2016, 463.68	1908, 2671.2	150, 120	3242,114	1796, 984
OctaCDD	0.0001	12420, 4595	9180, 10281	1170, 749	28844,289 6	11788 , 6708
2,3,7,8- TCDF	0.1	106.8, 9.61	84, 31.08	21, 14.7	100,8	16, 16
1,2,3,7,8- PeCDF	0.05	24.6, 4.67	6.6, 2.71	3.6, 1.51	30,10	8, 8
2,3,4,7,8- PeCDF	0.5	178.8, 25.03	65.4, 13.73	23.4, 12.87	807,108	720, 300
1,2,3,4,7,8- HxCDF	0.1	178.8, 30.40	43.8, 9.20	27.6, 11.04	293,20	208, 92
1,2,3,6,7,8- HxCDF	0.1	54, 3.78	27, 6.21	13.8, 5.52	261,17	176, 84
1,2,3,7,8,9- HxCDF	0.1	<0.05	<0.04	<0.04	NA	NA
2,3,4,6,7,8- HxCDF	0.1	55.8, 6.70	25.2, 6.80	9, 5.76	133,19	96, 52
1,2,3,4,6,7, 8-HpCDF	0.01	471, 117.75	176.4, 65.27	51.6, 22.19	523,55	240, 124
1,2,3,4,7,8, 9-HpCDF	0.01	39, 4.68	7.8, 5.07	3, 1.62	NA	4, 4
OctaCDF	0.0001	466.8, 107.36	195, 78.0	69.6, 37.58	49,10	12, 12

Chemical/ group	TEF	1978 (diet)^a	1984/5 (diet)^a	1994 (diet)^a	1988 (milk)^b	1993 (milk) ^a
		Mean, SD	Mean, SD	Mean, SD	Mean, SD	Mean, SD
		pg/d*	pg/d*	pg/d*	pg/kg- milk	pg/kg -milk
PCB-77	0.0001	NA	NA	NA	NA	452, 872
PCB-81	0.0001	NA	NA	NA	NA	NA
PCB-126	0.1	1350, 202.5	924, 221.76	378.6, 87.08	NA	3284, 1448
PCB-169	0.01	270, 54.0	181.2, 86.98	174, 214.02	NA	2320, 988
		ng/d*	ng/d*	ng/d*	ng/kg- milk	ng/kg -milk
PCB-105	0.0001	71.4, 13.57	70.2, 33.7	13.2, 5.54	NA	160, 80
PCB-114	0.0005	6.6, 0.92	11.4, 8.66	1.8, 1.35	NA	NA
PCB-118	0.0001	289.2, 43.38	247.2, 111.24	49.2, 15.25	1009,565	971.2, 456
PCB-123	0.0001	18.6, 3.91	15, 7.65	2.4, 0.89	NA	NA
PCB-156	0.0005	191.4, 63.16	27.6, 8.28	9, 2.79	NA	564, 236
PCB-157	0.0005	22.2, 6.44	4.8, 1.73	1.8, 0.72	NA	108, 48
PCB-167	0.0000 1	79.2, 22.18	11.4, 2.51	3.6, 1.01	NA	152, 64
PCB-189	0.0001	43.8, 13.14	2.4, 0.53	1.2, 0.31	NA	48.4, 48

^a (Liem et al., 2000); ^b (Albers et al., 1996), NA, not available; * Conversion from g-fat to kg-milk = 0.04 g-fat/g-milk*1000g/kg; Liem et al reported dietary intake estimates in units of mass/body weight/day. Therefore, we converted their estimates to units of mass/day by multiplying by the default 60 kg body weight used by Liem et al (Liem et al., 2000).

Liem et al (2000) reported dietary intake for three time-periods (see Table J.2-1)(Liem et al., 2000). Dietary intake estimates were based on concentrations of PCDD/Fs and PCBs measured in composite samples of 24-hr duplicate diets in the Dutch adult population in 1978, 1984-85, and 1994 and combined with individual consumption data collected in 1987-1988 (Albers et al., 1996) (briefly summarized previously) for approximately 6000 individuals from 2200 families over a 2-day period. In a separate study, these same investigators estimated dioxin and dioxin-like compounds in human milk fat collected in the period 1992-1993 from more than 80 women (Liem et al., 1995; Liem et al., 2000).

Liem et al (2000) observed a downward trend in estimated dietary intake of individual congeners of PCDDs PCDFs and PCBs in the Dutch population during

three intervals from 1978 to 1994 (see Table J.2-1)(Liem et al., 2000). A downward trend was also seen in a study of these toxicant levels in the diet and human milk of the German population from 1983 - 2003 (Furst, 2006; Wilhelm et al., 2007). However, about half of the mono-ortho PCBs did not show a similar linear decline. This pattern is consistent with observations made by Alcock and Jones (1996) who reported some evidence that the environmental load of PCDD/Fs increased in the 1960s, peaked around 1975 and then began to decline (Alcock et al., 1996).

OEHHA has derived lactational transfer coefficients for PCDD/Fs and dioxin-like PCBs from studies of exposure from global sources and by multiple pathways. The proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from that found from global sources. However, we assume that the estimate of transfer to milk from global sources, such as that derived from the Dutch studies, reasonably represents the transfer in persons from communities near Hot Spot facilities in California.

The Hot Spots program allows for reporting emissions of individual congeners of dioxins, furans and PCBs, when emissions are speciated. It also permits reporting of emissions as total dioxins and furans or PCBs. Speciation of emissions produces a more accurate (and lower) risk estimate. This is because unspciated emissions are assumed to be 2,3,7,8-TCDD, which has the highest potency factor among the dioxins and furans. Therefore, OEHHA has derived congener Tcos for individual PCBs and dioxins that can be used when emissions are speciated.

J.2.3 Mothers' Milk Transfer Coefficients (Tco) for PCDD/Fs and PCBs

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in adults estimated from dietary and occupational exposures. OEHHA estimated oral Tcos for these chemicals using estimates of body weight reported in Chapter 10 of this document, reference half-lives reported in Milbrath et al (2009) and the steady-state equation developed by Smith (1987) (Smith, 1987; Milbrath et al., 2009).

Milbrath et al (2009), in a systematic review of studies reporting half-lives in the human body, developed average human biological reference half-lives for 28 out of 29 dioxins and dioxin-like PCBs with OEHHA-recognized potency factors (see Table J-2-2) (Milbrath et al., 2009).

Table J.2-2: Half lives of PCDD/Fs and Dioxin-like PCB Congeners in Humans as Measured in Blood (Milbrath et al., 2009)

Chemical	N studies	Half life range (yrs)	Mean half life in adult (yrs)	Median half life in adult (yrs)	Study
TCDD	10	1.5 – 15.4	7.2	6.3	a
1,2,3,7,8-PeCDD	4	3.6 – 23.1	11.2	8.5	a
1,2,3,4,7,8-HxCDD	3	1.4 – 19.8	9.8	10.9	a
1,2,3,6,7,8-HxCDD	4	2.9 – 70	13.1	12	a
1,2,3,7,8,9-HxCDD	3	2.0 – 9.2	5.1	6.8	a
1,2,3,4,6,7,8-HpCDD	4	1.6 – 16.1	4.9	3.7	a
OctaCDD	4	1.8 - 26	6.7	5.7	a
2,3,7,8-TCDF	1	0.4	2.1	0.9	b
1,2,3,7,8-PeCDF	4	0.9-7.5	3.5	1.9	b
2,3,4,7,8- PeCDF	16	1.5-36	7	4.9	b
1,2,3,4,7,8-HxCDF	14	1.5-54	6.4	4.8	a
1,2,3,6,7,8-HxCDF	6	2.1-26	7.2	6	a
2,3,4,6,7,8-HxCDF	6	1.5-19.8	2.8	3.4	b
1,2,3,4,6,7,8-HpCDF	11	2.0-7.2	3.1	3	a
1,2,3,4,7,8,9-HpCDF	1	2.1-3.2	4.6	5.2	b
OctaCDF	1	0.2	1.4	1.6	b
PCB-77	2	0.1-5.02	0.1	0.1	c
PCB-81	-	-	0.7	0.73	c
PCB-126	3	1.2-11	1.6	2.7	c
PCB-169	3	5.2-10.4	7.3	10.4	c
PCB-105	4	0.56-7.0	2.4	2.4	c
PCB-114	2	7.4-31.7	10	25	c
PCB-118	10	0.82-33.7	3.8	1.6	c
PCB-123	2	5.3-15.3	7.4	12	c
PCB-156	7	1.62-100	16	5.35	c
PCB-157	2	13-26	18	20	c
PCB-167	2	8.7-35	12	12	c
PCB-189	2	16-166.7	22	41	c

^a (Flesch-Janys et al., 1996); ^b (van der Molen et al., 1996); ^c (Ogura, 2004)

Each reference half-life was derived from data on occupational exposures (Flesch-Janys et al., 1996; van der Molen et al., 1996) or dietary intake of the general population (Ogura, 2004). Note that mean half-lives vary by more than 2-fold among dioxin, 5-fold among furans and more than 100-fold among PCB congeners.

In an initial review of the literature, Milbrath et al (2009) reviewed evidence about factors that can affect elimination rates. Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Flesch-Janys et al., 1996; Milbrath et al., 2009). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce the maternal body burden of PCBs during 6 months of lactation (Niessen et al., 1984; Landrigan et al., 2002).

Half-lives derived from children would be less than that from older adults due, in part, to the effects of the growing body on estimates of blood concentrations. Models based on rat data demonstrate a linear relationship between increasing fat mass and half-life length at low body burdens, with the impact of adipose tissue on half-life becoming less important at high body burdens (Emond et al 2006). At high body burdens, dioxins are known to up-regulate the enzymes responsible for their own elimination. Human data suggest that the serum concentration of TCDD where this transition occurs is 700 pg/g and 1,000 – 3,000 pg/g for PCDFs (Kerger et al 2006, Leung et al 2005). Therefore, investigators selected a subset of data based on the following criteria:

- blood serum concentrations of PCDD/Fs were less than 700 pg /g blood lipid total toxic equivalents (TEQs) at the time of sampling
- subjects were adults
- measurements were not reported as inaccurate in later studies

Milbrath et al selected the reference values to represent a 40- to 50-year-old adult with blood dioxin concentrations in the range where fat drives the rate of elimination (i.e. at lower body burdens). In addition, Milbrath rejected half-lives longer than 25 years if the original study calculated half-lives assuming steady-state conditions.

For the retained subset, the investigators calculated the mean and range of half-lives to establish a representative set of half-lives for each congener in a moderately exposed adult (Milbrath et al., 2009). They also adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans (Milbrath et al., 2009).

A generally accepted approach to estimating the concentration of a lipophilic chemical in milk is outlined by Smith (1987). This approach is based on average

maternal daily intake, an estimate of the half-life ($t_{1/2}$) of PCDDs/PCDFs and PCBs and body weight-normalized (BW) proportionality factors. The chemical concentration in breast milk can be calculated by equation J-4:

$$C_m = (E_{mi})(t_{1/2})(f_1)(f_3)/(f_2)(0.693) \quad \text{Eq. J-4}$$

C_m = chemical concentration in milk (mg/kg milk)

E_{mi} = average daily maternal intake of contaminant (mg/kg-BW/day)

$t_{1/2}$ = biological half-life (days)

f_1 = proportion of chemical in mother that partitions into fat (e.g. 0.8)

f_2 = proportion of mother's body weight that is fat (e.g. 0.33 = kg-fat/kg-BW)

f_3 = proportion of breast milk that is fat (e.g. 0.04 = kg-fat/kg-milk)

Smith's approach requires an estimate of the biological half-life of PCBs and PCDDs/PCDFs in the adult human and is restricted to poorly metabolized, lipophilic chemicals that act predominantly by partitioning into the fat component and quickly reaching equilibrium in each body tissue (including breast milk).

Because of Milbrath's approach, T_{co} -estimates for dioxins, furans and dioxin-like PCBs apply the following conservative assumptions regarding factors that affect elimination rates:

- lower enzyme induction based on nonsmokers with a body burden below 700 ppt in the blood
- adult age
- no recent history of breast-feeding
- body fat estimates based on older adults

Transfer coefficients (Ng, 1982) are ideally calculated from the concentration of contaminant in milk following relatively constant long-term exposure that approximates steady state conditions. Because Smith's equation is linear, it can be rearranged to solve ratio of the chemical concentration in milk to the chemical taken into the body per day, which is the transfer coefficient (Equation J-5).

$$T_{co} = C_m/(C_f)(I) \quad \text{Eq J-5}$$

T_{co} is the transfer coefficient (day/kg or day/liter)

C_m = measured chemical concentration in milk ($\mu\text{g/kg}$ or mg/liter milk)

C_f = measured chemical concentration in exposure media (e.g. food) ($\mu\text{g/kg}$ food)

I = reported daily intake of exposure media (kg/day of food)

The following equation (Eq-J-6) is equation Eq J-5 substituted into equation Eq J-4 and rearranged to solve for T_{co} .

$$Tco = (t_{1/2})(f1)(f3)/(BW)(f2)(0.693)$$

Eq J-6

Note that Emi in equation J-4 = (Cf)(I)/BW with units of mg/kg-BW/day. BW is the average adult body weight of the mother (kg).

Transfer coefficients (Tcos) in Table J.2-3 (column-2) combine milk data (milk concentration of PCDD/Fs and PCBs) with dietary intake estimates listed in Table J.2-1. OEHA derived individual Tcos from data presented in (Liem et al., 1995; Albers et al., 1996; Liem et al., 2000). Because the median is a reasonable estimate of the geometric mean in skewed distributions, Tcos were derived from median half-lives listed in column-5 of Table J.2-2. Tcos range from less than one to more than ten d/kg-milk among dioxins and furan and less than two to more than 20 d/kg-milk among dioxin-like compounds.

Table J.2-3: Arithmetic mean Transfer Coefficients (Tcos) for Individual PCDD/F and PCB Congeners Measured in Human Milk and Dietary Intake from a Dutch Population (d/kg-milk) Compared to the Median and Geometric Mean Tcos Derived from Reference Half-lives ($t_{1/2}$) and Equation J-6

Chemical/group	Tcos (GM) based on slope factors	Tco based on median reference half life (Milbrath et al 2007)	Tco based on $t_{1/2}$ GM*	Tco based on $t_{1/2}$ GSD	Tco based on $t_{1/2}$ LCL	Tco based on $t_{1/2}$ UCL
2,3,7,8-TCDD	49.62	5.36	4.02	2.76	2.14	7.53
1,2,3,7,8-PeCDD	8.76	7.24	6.53	2.16	3.07	13.90
1,2,3,4,7,8-HxCDD	0.98	9.28	5.60	3.41	1.40	22.48
1,2,3,6,7,8-HxCDD	11.02	10.21	3.27	4.20	0.80	13.32
1,2,3,7,8,9-HxCDD	4.89	5.79	3.32	1.91	1.60	6.88
1,2,3,4,6,7,8-HpCDD	2.88	3.15	1.96	2.74	0.73	5.26
OctaCDD	5.54	4.85	2.29	3.25	0.72	7.28
2,3,7,8-TCDF	3.18	0.77	1.76	1.36	0.96	3.23
1,2,3,7,8-PeCDF	3.43	1.62	1.91	2.49	0.78	4.68
2,3,4,7,8- PeCDF	2.77	4.17	1.78	4.24	0.88	3.62
1,2,3,4,7,8-HxCDF	2.16	4.09	0.99	5.29	0.41	2.38
1,2,3,6,7,8-HxCDF	7.89	5.11	2.64	3.01	1.09	6.39
1,2,3,7,8,9-HxCDF	NA	NA	NA	NA	NA	NA
2,3,4,6,7,8-HxCDF	3.18	2.89	0.55	3.18	0.22	1.39
1,2,3,4,6,7,8-HpCDF	2.40	2.55	1.82	1.63	1.36	2.44
1,2,3,4,7,8,9-HpCDF	NA	4.43	3.63	1.34	2.06	6.42
OctaCDF	0.32	1.36	0.99	2.83	0.13	7.55
PCB-77	NA	NA	0.06	6.38	0.004	0.72
PCB-81	NA	NA	0.38	1.35	0.248	0.57

Chemical/group	Tcos (GM) based on slope factors	Tco based on median reference half life (Milbrath et al 2007)	Tco based on t1/2 GM*	Tco based on t1/2 GSD	Tco based on t1/2 LCL	Tco based on t1/2 UCL
PCB-126	NA	2.30	0.34	2.61	0.11	1.01
PCB-169	NA	8.85	5.60	1.27	4.28	7.32
PCB-105	NA	2.04	1.07	3.02	0.36	3.16
PCB-114	NA	2.04	2.74	3.11	0.57	13.20
PCB-118	0.01	1.36	0.55	6.17	0.18	1.70
PCB-123	NA	1.36	2.93	2.63	0.77	11.18
PCB-156	NA	4.55	3.23	7.10	0.76	13.81
PCB-157	NA	17.02	14.10	1.21	10.84	18.34
PCB-167	NA	10.21	5.93	1.76	2.70	13.00
PCB-189	NA	34.90	4.23	2.77	1.03	17.33

slope factors obtained from the longest interval between measures of diet (1978-1994) and milk (1988-1993) in the Dutch population; * GM, geometric mean, GSD, geometric standard deviation derived from natural log of three half-life values, low, high and median reported in Milbrath et al 2007 (Milbrath et al., 2009) LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA evaluated the relationship between Tcos predicted by Equation J-6 (column 3) using median reference half-lives and those derived from slope factors (column 2). Briefly, slope factors were calculated by taking the difference between cross-sectional dietary intake estimates taken in 1978 and 1994 and the difference between cross-sectional human milk concentrations taken in 1988 and 1993 from the Dutch population. Most Tcos derived from reference half-lives compare reasonably well with those derived from slope factors.

In columns 4-7 of Table J.2-3 the GM, GSD and 95%CLs of transfer coefficients (Tcos) for individual dioxins and dioxin-like congeners are derived from equation J-6 and geometric distribution estimates and 95% confidence intervals of half-lives provided in (Milbrath et al., 2009).

A Random-effects model derived summary estimates shown in Table J.2-4 from individual summary estimates shown in columns 4-7 of Table J.2-3.

Table J.2-4: Tco Estimates Stratified by Dioxin, Furan and Dioxin-like PCB Congeners (mean, 95%CI from Random-effects Model)

Chemical group	N congeners	Tco	LCL	UCL
PCDDs - oral	7	3.7	2.68	5.23
PCDFs - oral	9	1.8	1.27	2.43
Dioxin-like PCBs - oral	12	1.7	0.69	4.40

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA believes that a Random-effects model is appropriate because OEHHA assumes that the compounds found in exposure studies are a subgroup from a population of congeners in each subgroup (i.e., dioxins and dioxin-like compounds). Random-effects models assume there are multiple central estimates and incorporate a between-compound estimate of error as well as a within-compound estimate of error in the model. In contrast, a Fixed-effects model assumes observations scatter about one central estimate (Kleinbaum, 1988).

J.2.4 Carryover Rate

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of dioxins and dioxin-like compounds in the mother's body occurs but varies by more than 100-fold among individual compounds (based on Tcos derived from equation J-6).

Carryover rate, a term commonly used in the dairy literature (McLachlan et al., 1990) is defined as the daily output of dioxins and dioxin-like compounds in mother's milk ($\mu\text{g}/\text{day}$) over the daily intake of dioxins and dioxin-like compounds ($\mu\text{g}/\text{day}$). This rate is estimated by multiplying a dioxins and dioxin-like Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, a dioxins and dioxin-like Tco is the carryover rate for a typical 60 kg woman.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. With an average dioxin Tco of 3.7 d/kg, daily intake of dioxins, or 370% of the daily intake from ingested sources, transfers to mother's milk. This high transfer-value suggests that some accumulation or concentrating of carcinogenic dioxins and dioxin-like compounds occur in the mother's body. Oral Tcos less than one d/kg (e.g., 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggests that some metabolism occurs in the mother's body.

J.3 Mothers' Milk Transfer Coefficients for PAHs

The polycyclic aromatic hydrocarbons (PAHs), a family of hundreds of different chemicals, are characterized by fused multiple ring structures. These compounds are formed during incomplete combustion of organic substances (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Thus, PAHs are ubiquitous in the environment and humans are likely to be exposed to these compounds on a daily basis. PAHs are a common pollutant emitted from Hot Spots facilities and are evaluated under the program.

Only a small number of the PAHs have undergone toxicological testing for cancer and/or noncancer health effects. PAHs with cancer potency factors are the only ones that can be evaluated for cancer risk using risk assessment. However, PAHs that lack cancer potency factors have been measured in various studies and can serve as a useful surrogate for PAHs with cancer potency factors because of their physical-chemical similarity to PAHs with cancer potency factors.

Less than 30 specific PAHs are measured consistently in biological samples or in exposure studies. For example, Table J.3-1 lists commonly detectable PAHs in food and the environment (Phillips, 1999). In one analysis, pyrene and fluoranthene together accounted for half of the measured PAH levels in the diet (Phillips, 1999). Table J.3-1 includes nine PAHs that have cancer potency factors and are recognized by OEHHHA as presenting a carcinogenic risk to humans (OEHHHA, 2009).

Table J.3-1: PAHs with and without Cancer Potency Factors Commonly Measured in Food (Phillips, 1999)

PAHs without Cancer Potency Factors	PAHs with Cancer Potency Factors
Benzo[ghi]perylene	Dibenz[a,h]anthracene
Fluoranthene	Indeno[1,2,3-cd]pyrene
Pyrene	Benzo[a]pyrene
Phenanthrene	Benzo[k]fluoranthene
Anthracene	Chrysene
Fluorene	Benzo[b]fluoranthene
Acenaphthylene	Benz[a]anthracene
Acenaphthene	Naphthalene
Benzo[b]naphtho[2,1-d]thiophene	Benzo[j]fluoranthene
Benzo[ghi]fluoranthene	
Cyclopenta[cd]pyrene	
Triphenylene	
Perylene	
Benzo[e]pyrene	
Dibenz[a,j]anthracene	
Anthanthrene	
Coronene	

Few investigators have attempted to correlate PAH exposure from contaminated food and ambient air with PAH concentrations in human biological samples such as the blood or mother's milk. This is likely due to insensitive limits of detection for PAHs yielding few positive measurements, possibly due to the rapid and extensive metabolism of PAHs in mammals (West and Horton, 1976; Hecht et al., 1979; Bowes and Renwick, 1986).

This extensive metabolism often results in low or immeasurable concentrations of PAHs in mother's milk and blood (e.g. (Kim et al., 2008)). Nevertheless, emissions of PAHs from stationary sources are common and the increased sensitivity of infants to carcinogens necessitates looking into development of mother's milk transfer factors (Tco) for carcinogenic PAHs.

Four studies have measured PAHs in mother's milk of smokers and non-smokers (see Table J.3-2). The 16 PAHs reported in these studies are among the most common PAHs released into the environment and found in biological samples (Phillips, 1999; Ramesh et al., 2004).

TABLE J.3-2: Measured Concentrations ($\mu\text{g/kg-milk}$) of PAHs in Human Milk

Chemical / chemical group	Urban smokers (Italy) n=11 ^a (Zanieri et al., 2007)	Urban non-smokers (Italy) n=10 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=11 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=10 (Del Bubba et al., 2005)	Non-smokers (USA) n=12 (Kim et al., 2008)	Un-known (Japan) n=51 (Kishikawa et al., 2003)
PAHs with Cancer Potency Factors AM, SD						
Naphthalene	10.54, 6.08	6.83, 2.18	4.42, 1.17	4.70, 2.44	NA ^d	NA
Chrysene	0.90, 2.09	0.59, 0.94	<0.018	<0.018	-- ^c	0.06, 0.08
Benzo[a]anthracene	0.98, 1.47	0.61, 0.94	0.07, 0.16	0.974, 1.82	--	0.004, 0.01
Benzo[b]fluoranthene	0.53, 1.24	0.55, 0.80	<0.019	0.560, 1.39	--	0.41, 0.26
Benzo[k]fluoranthene	0.13, 0.30	<0.018	<0.018	0.114, 0.343	--	0.01, 0.01
Benzo[a]pyrene	0.52, 0.65	<0.018	<0.018	<0.018	--	0.002, 0.003
Dibenzo[a,h]anthracene	1.33, 3.33	<0.014	<0.014	<0.014	--	0.01, 0.01
Indeno[1,2,3-c,d]pyrene	0.42, 0.94	<0.011	<0.011	<0.011	--	0.003, 0.01
Sum	15.35	8.58	4.5	6.4	--	0.5
PAHs without Cancer Potency Factors AM, SD						
Anthracene	0.16, 0.45	0.71, 1.57	0.21, 0.56	0.616, 1.58	-- ^c	0.01, 0.01
Acenaphthylene	7.73, 11.95	9.09, 3.08	4.11, 3.62	6.95, 4.18	NA ^d	NA
Phenanthrene	3.67, 2.39	0.97, 0.51	0.64, 0.58	0.553, 0.493	0.49, 0.44	0.25, 0.16

Chemical / chemical group	Urban smokers (Italy) n=11 ^a (Zanieri et al., 2007)	Urban non-smokers (Italy) n=10 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=11 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=10 (Del Bubba et al., 2005)	Non-smokers (USA) n=12 (Kim et al., 2008)	Unknown (Japan) n=51 (Kishikawa et al., 2003)
Fluorene	5.13, 9.45	1.50, 1.60	0.06, 0.21	1.06, 1.70	0.13, 0.13	NA
Acenaphthene	10.55, 17.73	3.12, 1.79	1.37, 1.31	2.72, 1.69	NA	NA
Pyrene	1.03, 1.25	1.40, 3.01	0.21, 0.30	0.620, 1.64	0.05, 0.04	0.02, 0.05
Fluoranthene	2.86, 2.60	0.54, 0.76	0.53, 1.03	0.250, 0.441	0.06, 0.05	0.02, 0.03
Benzo[g,h,i] perylene	1.51, 2.24	<0.018	<0.018	<0.018	--	--
Sum	32.64	17.33	7.13	12.8	0.73	0.3

^a group includes one rural smoker; ^b values below detection limits were treated as zero in estimates of the mean; ^c – indicates all measurements were below the detection limits; ^d not assessed; (Kishikawa et al., 2003; Del Bubba et al., 2005; Zanieri et al., 2007; Kim et al., 2008) µg, microgram; kg, kilogram; n, number of samples; AM, Arithmetic Mean; SD, Standard Deviation

In this section, we estimated Tcos for PAHs with and without cancer potency factors. Additionally, none of the PAHs has a chronic Reference Exposure Level (REL) value associated with them. PAHs without cancer potency factors (other) are included because they:

- have structures similar to carcinogenic PAHs and are thus suitable as surrogate compounds
- are frequently measured in exposure studies
- produce measurements at detectable levels

In Table J.3-2, the sum of carcinogenic PAHs in human milk of Italian women are about 2-fold lower than the sum of other PAHs.

Because of their similarities in structure, the Tcos developed from other abundant PAHs are expected to compare reasonably well with the Tcos developed for less abundant carcinogenic PAHs.

J.3.1 Inhalation Biotransfer of PAHs to Mother's Milk

Biotransfer of PAHs to breast milk via the mother's inhalation pathway must be considered separately from biotransfer of PAHs to breast milk from the mother's

oral route. PAHs will show a different pattern of metabolism depending on the route of exposure because of first pass metabolism in the liver from oral exposure, different rates and patterns of metabolism in the lung, and other factors. Smoking cigarettes represents a significant source of PAHs resulting in measurable levels of PAHs in mother's milk. Therefore, OEHHA chose a study that measured PAH concentrations in breast milk in smoking women and nonsmoking women to estimate inhalation Tcos for PAHs.

Of the four studies listed in Table J.3-2, the Italian study by Zanieri et al. (2007) allowed correlation of PAH intake via chronic smoking with PAH levels found in human milk (Zanieri et al., 2007). These investigators reported individual PAH concentrations in the milk of urban smoking and nonsmoking mothers, and in rural smoking and nonsmoking mothers.

Zanieri et al (2007) had obtained self-reported smoking habits (an arithmetic average of 5.4 cigarettes smoked per day) but not the daily dose of PAHs due to smoking (Zanieri et al., 2007). Therefore, OEHHA estimated daily PAH doses using published estimates of the amounts of PAHs a smoker voluntarily consumes during smoking per cigarette from simulated cigarette smoking studies. (Ding et al., 2005) measured the amount of 14 individual PAHs that would be inhaled because of smoking major U.S. cigarette brands (Table J.3-3). Two other simulated smoking studies were included that estimated the inhaled amounts of two additional PAHs not covered in the Ding study (Gmeiner et al., 1997; Forehand et al., 2000).

Table J.3-3: Summary Estimates of Polycyclic Aromatic Hydrocarbons (PAHs) Intake from Cigarettes ($\mu\text{g}/\text{cigarette}$)

PAH	Ding et al (n=5)	Ding et al (n=50)	Ding et al (n=5)	Gmeiner et al (n=3)	Forehand et al (n=4)	Pooled
With Cancer Potency Factors	1 [#] AM, SD ¹	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Naphthalene	0.3503 , 0.021	0.192, 0.044	0.407, 0.187	0.236, 0.019	0.362, 0.011	0.292, 0.087
Chrysene	0.0157 , 0.0003	0.0197, 0.0024	0.0314, 0.0028	0.0218, 0.0009	0.0112, 0.0003	0.015, 0.0017
Benzo[a]anthracene	0.0134 , 0.0007	0.0165, 0.0015	0.0226, 0.0025	0.0132, 0.0005	0.014, 0.0004	0.015, 0.0014
Benzo[b]fluoranthene	0.0094 , 0.003	0.0106, 0.0013	0.0183, 0.0024	0.0086, 0.0003	0.0112, 0.0003	0.010, 0.0012

PAH	Ding et al (n=5)	Ding et al (n=50)	Ding et al (n=5)	Gmeiner et al (n=3)	Forehand et al (n=4)	Pooled
With Cancer Potency Factors	1[#] AM, SD¹	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Benzo[k]fluoranthene	0.0015, 0.00014	0.0019, 0.00029	0.0039, 0.00070	0.0015, 0.00008	NA	0.0020, 0.0004
Benzo[a]pyrene	0.0103, 0.00041	0.011, 0.00077	0.0147, 0.00118	0.0079, 0.00024	0.0076, 0.00023	0.0092, 0.00067
Dibenzo[a,h]anthracene	NA	NA	NA	0.0006, 0.00013	0.0023, 0.00021	0.0023, 0.00017
Indeno[1,2,3-c,d]pyrene	NA	NA	NA	0.0035, 0.00039	NA	0.0035, 0.00039
Without Cancer Potency Factors	1 AM, SD	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Anthracene	0.0749, 0.0052	0.0698, 0.0084	0.074, 0.0089	0.0381, 0.0023	0.0358, 0.0011	0.043, 0.0060
Acenaphthylene	0.1169, 0.0082	0.0883, 0.0097	0.153, 0.0306	0.0504, 0.0040	NA	0.083, 0.0167
Phenanthrene	0.1348, 0.0054	0.1452, 0.0131	0.144, 0.0144	0.11, 0.0033	0.1477, 0.0044	0.134, 0.0094
Fluorene	0.2175, 0.0087	0.1563, 0.0188	0.257, 0.0257	0.119, 0.0048	0.239, 0.0048	0.184, 0.0151
Acenaphthene	0.0848, 0.0025	0.0513, 0.0072	0.088, 0.0167	0.0253, 0.0013	NA	0.062, 0.0092
Pyrene	0.0486, 0.0029	0.0495, 0.0069	0.077, 0.0231	0.0332, 0.0017	0.0321, 0.0010	0.036, 0.0109
Fluoranthene	0.0744, 0.0037	0.063, 0.0107	0.101, 0.0121	0.0462, 0.0018	0.0516, 0.0026	0.056, 0.0076

PAH	Ding et al (n=5)	Ding et al (n=50)	Ding et al (n=5)	Gmeiner et al (n=3)	Forehand et al (n=4)	Pooled
With Cancer Potency Factors	1[#] AM, SD¹	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Benzo[g,h,i] perylene	NA	NA	NA	0.0025, 0.00030	0.0023, 0.00018	0.0023, 0.0002 5

¹AM arithmetic mean,, SD standard deviation ; #, Experiment number listed in the study reference by the first author in row one of columns two through six in the table, (Gmeiner et al., 1997; Forehand et al., 2000; Ding et al., 2005)

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers by Zanieri et al. (2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1:

$$Tco_{hmi} = Cm_i / (C_{cig_i} \times I_{cig/day} \times f_{smoke}) \quad \text{Eq. J-7}$$

where:

Cm_i = adjusted geometric average ith PAH concentration due to smoking (µg per kg milk as wet weight)

C_{cig_i} = geometric average dose of the ith PAH per cigarette (µg/cigarette averaged across experiments)

$I_{cig/day}$ = geometric average number of cigarettes smoked (4.75 cigarettes/day)

f_{smoke} = adjustment for under-reporting of smoking frequency (2)

Cm_i is the adjusted geometric average of the ith PAH in whole milk due to smoking. OEHHA obtained these estimates by converting arithmetic estimates to geometric estimates of the mean and standard deviation and subtracting the GM concentration in the milk of primarily urban nonsmokers from the GM concentration in the milk of urban smokers. This adjustment accounts for oral intake of PAHs from dietary sources and inhalation of PAHs in urban air from combustion sources other than cigarettes. Implicit in this adjustment is the assumption by OEHHA that oral intake and exposure to other airborne PAHs is similar between smokers and nonsmokers who participated in the Zanieri study.

OEHHA also included a 2-fold smoking habit adjustment-factor (f_{smoke}) in Eq. J-7 based on published data to account for the recognized tendency of smokers to under-report their smoking habits. The studies examined the accuracy of self-reported smoking habits among pregnant women and parents with small children (Marbury et al., 1993; Graham and Owen, 2003). They measured airborne nicotine in the smoker's breathing zone and obtained the number of cigarettes smoked per day by each smoker. The data presented in Figure (1) of Marbury et

al suggest that mothers under-reported their smoking rate by 50% (Marbury et al., 1993).

Tables J.3-4 presents the Tcos for cancer and noncancer PAHs calculated using Eq. J-7. However, Zanieri and Del Bubba did not find measurable levels of some PAHs, particularly PAHs with 5 or 6 carbon rings, in milk from nonsmokers. In these cases, the concentration representing half the limit of detection (between 0.006-0.014 µg/kg) was used as the background concentration of the PAH in mother's milk.

There are two main limitations in the data provided in Table J.3-4. For some PAHs, no individual Tco was calculated because the concentration of the individual PAH was higher in mother's milk of nonsmokers than in smokers. For example, in column two of Table J.3-4, mother's milk benzo[b]fluoranthene, pyrene and anthracene have negative concentration values.

These discrepancies could be due to the natural variation in the ability of individuals to transfer inhaled PAHs to milk, or as Zaneiri et al. suggested, a result of greater exposure to certain PAHs in some foods compared to cigarette smoke. The small sample sets (n=11 for each group of smokers and nonsmokers) in the Zanieri study are less likely to represent the true mean in the study population and magnify the large variation in this biological response.

Additional uncertainties in the use of smokers to estimate PAH transfer coefficients include that fact that lung metabolism may be different in smokers because of the much higher doses of PAHs that smokers receive relative to those only exposed in ambient pollution. Cytochrome P-450 enzymes are known to be induced when exposure is greater and therefore metabolism could be proportionately greater in smokers. In addition, at higher dose levels some enzyme systems may become saturated which could alter the pattern of metabolism.

However, smokers are the best population for estimating PAH Tcos because the inhalation dose can be separated from background inhalation and dietary exposure, and the inhalation dose from the cigarettes can be estimated. OEHHHA requested raw data from the investigators for individual women in the study, but unfortunately, only the summary statistics from the published paper were available to us.

Table J.3-4: Inhalation Transfer Coefficients (Tcos) for Individual PAHs with and without Potency factors from Geometric Mean and Standard Deviation Estimates (GM, GSD) of Human Milk (Cm) and Intake from Cigarettes (C_{cig}) (d/kg-milk)

PAH (no. of rings) ^a	Adjusted Cm (µg/kg wet wt.)	C _{cig} (µg/cig)	Inhalation Tco ^b (d/kg)
With Cancer Potency Factors	GM, GSD	GM, GSD	GM, GSD
Naphthalene (2)	2.78, 1.63	0.2798, 1.34	1, 2.66
Chrysene (4)	0.04, 5.34	0.0149, 1.12	0.28, 8.11
Benzo[a]anthracene (4)	0.20, 4.31	0.0149, 1.1	1.4, 6.52
Benzo[b]fluoranthene (5)	-0.09, 5.01	0.0099, 1.13	NA ^c
Benzo[k]fluoranthene (5)	0.05, 2.95	0.002, 1.22	0.26, 4.6
Benzo[a]pyrene (5)	0.26, 2.29	0.0092, 1.08	2.97, 3.45
Dibenzo[a,h]anthracene (5)	0.46, 3.85	0.0023, 1.08	2.11, 5.81
Indeno[1,2,3-c,d]pyrene (6)	0.16, 3.65	0.0035, 1.12	4.81, 5.54
Without Cancer Potency Factors	GM, GSD	GM, GSD	GM, GSD
Anthracene (3)	-0.22, 6.29	0.0426, 1.15	NA
Acenaphthylene (3)	-4.56, 2.9	0.0814, 1.22	NA
Phenanthrene (3)	2.00, 1.94	0.0035, 1.07	1.57, 2.92
Fluorene (3)	1.31, 4.1	0.1336, 1.09	0.75, 6.19
Acenaphthene (3)	2.48, 3.26	0.0613, 1.16	4.21, 5
Pyrene (4)	0.04, 4.57	0.0345, 1.34	0.12, 7.48
Fluoranthene (4)	1.63, 3.29	0.0555, 1.14	3.06, 5.02
Benzo[g,h,i]perylene (6)	0.77, 2.72	0.0023, 1.11	35.24, 4.13

^a no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat); ^b Sum of each PAH found in mother's milk microgram per kilogram (µg/kg) over the sum of the daily intake (µg/day) of the same PAH x 4.75 cigarettes/day x an adjustment factor of 2; ^c NA, not available because the concentration of PAH in mother's milk of smokers was lower than the concentration in nonsmokers, so an individual Tco could be calculated

Tco values for carcinogenic PAHs in Table J.3-4 are determined for all available PAHs and included in a summary estimate (see Table J.3-7 near the end of this section).

Unlike the other PAHs with cancer potency factors, naphthalene is not considered a multipathway chemical under the Hot Spots program because it is regarded as a gas, and therefore not subject to appreciable deposition onto soil,

etc. Naphthalene was included in this analysis because this PAH constitutes a large proportion of the total mass of PAHs inhaled. Among the carcinogenic PAHs in Table J.3-4, naphthalene predominates in both mainstream smoke (63% of total carcinogenic PAHs) and in mother's milk (56% of total carcinogenic PAHs). Naphthalene is also the only PAH that is considered a gas, and therefore, its physical properties are different from other larger PAHs that are semi-volatile or exist primarily as a solid. In spite of these differences, the summary estimate did not change when naphthalene was excluded in the analysis (summary Tco = 1.55 versus 1.60).

Due to few measurable levels of carcinogenic PAHs in milk samples, there is more uncertainty in the carcinogenic PAH Tco compared to the PAH Tco for PAHs without cancer potency values. Nevertheless, summary estimates for PAH Tcos from inhaled sources differ by less than a factor of two (Tco for carcinogens, 1.2 versus Tco without cancer potency values, 2.06) suggesting that there may be no systematic difference between these two groups of chemicals. Therefore, OEHHA combined individual Tcos for PAHs from both groups into an overall inhalation Tco (see Table J.3-7 and Figure J.3-1 at the end of this section of the Appendix). In Figure J.3-1, the top seven estimates of inhalation Tcos are carcinogenic PAHs and the bottom six estimates are PAHs without cancer potency values.

The combined estimate is the summary of all 13 PAH estimates combined using a Random-effects model. OEHHA assumes that the PAHs found in exposure studies are a subgroup from a population of PAHs. Random-effects models assume there are multiple central estimates and incorporate a between-PAH estimate of error as well as a within-PAH estimate of error. In contrast, a Fixed-effects model assumes observations scatter about one central estimate (Kleinbaum, 1988).

OEHHA recommends using the inhalation Tco based on the summary estimates provided in Table J.3-7 rather than using the individual PAH Tcos values provided in Table J.3-4, to assess transfer of individual inhaled PAHs to mother's milk. There are a high number of non-detects and small sample sizes in these data. The estimation of PAH Tco values with this method might be improved with more sensitive methods for measurement of breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds.

The key assumption underlying the development of these Tcos is that the variability in individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

J.3.2 Oral Biotransfer of PAHs to Mother's Milk

Diet is the largest contributor by pathway to total PAH intake from ubiquitous background sources for the general public and other situations where airborne levels are not remarkably high (Lioy et al., 1988). In a risk assessment of a reference nonsmoking male, a mean total PAH intake of 3.12 µg/d was estimated of which dietary intake was 96.2%, air 1.6%, water 0.2% and soil 0.4% (Menzie et al., 1992; Ramesh et al., 2004). Inhalation, soil ingestion and homegrown produce pathways can be important when considering total dose from a single stationary source. PAHs contaminate homegrown produce and soil through direct deposition. Milk and meat from home-raised animals or commercial sources would be less of a contributor because many PAHs are highly metabolized by these animals following intake from contaminated pastures and soil.

There are no studies available that relate PAH dietary intake directly to mother's milk concentrations for these compounds, although studies of PAH dietary intake have been performed in several countries. Therefore, the PAH biotransfer efficiency to mother's milk from food was calculated using PAH dietary intake data and mother's milk PAH data from separate studies. OEHHA recognizes the uncertainty in this approach but it appears to be the best currently available. Table J.3-5 shows the daily dietary intake of carcinogenic PAHs from published studies of European residents.

Table J.3-5: Summary Estimates of PAHs with and without Cancer Potency Factors Dietary Intake (µg/day)

PAH (no. of rings ^a)	Italian Lodovici et al (1995) Adults	Dutch De Vos et al. (1990) ^c Adult males	Spanis h Marti- Cid et al. (2008) Adults	Spanish Falco et al. (2003) Adults	U.K. Dennis et al. (1983) Adults
With Cancer Potency Factors	AM^b, SD	AM*	AM*	AM, SD	AM*
Naphthalene (2)	NA ^d	NA	1.846	0.823, 0.056	NA
Chrysene (4)	0.84, 0.0131	0.86 – 1.53	0.204	0.564, 0.037	0.5
Benzo[a]anthracene (4)	0.47, 0.0093	0.2 – 0.36	0.139	0.310, 0.021	0.22
Benzo[b]fluoranthene (5)	0.17, 0.0101	0.31 – 0.36	0.137	0.188, 0.014	0.18
Benzo[k]fluoranthene (5)	0.06, 0.0043	0.1 – 0.14	0.086	0.094, 0.006	0.06
Benzo[a]pyrene (5)	0.13, 0.0003	0.12 – 0.29	0.083	0.113, 0.008	0.25

PAH (no. of rings^a)	Italian Lodovici et al (1995) Adults	Dutch De Vos et al. (1990) ^c Adult males	Spanish h Martí- Cid et al. (2008) Adults	Spanish Falco et al. (2003) Adults	U.K. Dennis et al. (1983) Adults
Dibenzo[a,h]anthracene (5)	0.01, 0.0026	NDd ^e	0.084	0.048, 0.003	0.03
Indeno[1,2,3-c,d]pyrene (6)	ND	0.08 – 0.46	0.102	0.045, 0.003	ND
Without Cancer Potency Factors	AM, SD	AM*	AM*	AM, SD	AM*
Anthracene (3)	NA	0.03 – 0.64	0.428	0.088, 0.006	NA
Acenaphthylene (3)	NA	NA	0.354	0.402, 0.026	NA
Phenanthrene (3)	NA	NA	3.568	2.062, 0.150	NA
Fluorene (3)	NA	NA	0.934	0.206, 0.017	NA
Acenaphthene (3)	NA	NA	0.368	0.071, 0.005	NA
Pyrene (4)	0.19, 0.0043	NA	1.084	1.273, 0.092	1.09
Fluoranthene (4)	1.03, 0.0106	0.99 – 1.66	1.446	0.848, 0.062	0.99
Benzo[g,h,i]perylene (6)	0.20, 0.0009	0.2 – 0.36	0.112	0.214, 0.017	0.21

^a no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat); ^b Arithmetic mean (AM), Standard Deviation (SD); ^c The Dutch dietary intakes were presented as the range of lower bound values (calculated by taking values below the detection limit to be zero) to upper bound values (calculated by taking values below the detection limit to be equal to the limit) ^d NA, Not available; ^e Nd, Not determined; * no measure of variance was reported (Dennis et al., 1983a; Dennis et al., 1983b; De Vos et al., 1990; Lodovici et al., 1995; Falcó et al., 2003; Martí-Cid et al., 2008)

Regional preferences, ethnicity, and individual dietary preferences will influence the amount of PAHs ingested with food. In addition, there were differences among the intake studies in the number and type of PAHs investigated in foods. Even though dietary habits and PAH analysis methods can result in different levels of PAH intake, the total dietary intakes of PAHs in each of five studies in Table J.3-5 were generally within an order of magnitude of each other.

Mother's milk PAH concentration data from nonsmoking rural lactating women living in Italy by Zaneiri et al. (2007) and Del Bubba et al. (2005) were pooled and paired with estimates of PAH dietary intake in the Italian population (Lodovici et al., 1995; Del Bubba et al., 2005; Zanieri et al., 2007). The mother's milk PAH data are described above in the PAH inhalation biotransfer section. The study by Del Bubba et al. is a similar study by the same research group that includes additional participants from rural areas. The use of nonsmoking rural women should reduce confounding contributions from the inhalation pathway. Airborne concentrations of PAHs tend to be higher in urban areas due to mobile sources.

Based on the estimated intake of the same measured PAHs in dietary studies and the PAHs found in breast milk from nonsmoking mothers (Del Bubba et al., 2005; Zanieri et al., 2007), OEHHA was able to estimate transfer coefficients (Tco) by Equation J-8, a version of Equation J-1:

$$Tco_{hmoi} = C_{m_{oi}} / (D_{oi}) \quad \text{Eq. J-8}$$

where:

$C_{m_{oi}}$ = geometric average ith PAH concentration in mother's milk (μg per kg milk as wet weight)

D_{oi} = geometric average dose of the ith PAH per day from dietary sources ($\mu\text{g}/\text{day}$)

$C_{m_{oi}}$ is the geometric average of the ith PAH in whole milk from nonsmoking, rural dwelling women. OEHHA obtained estimates of GM and GSD by pooling and converting arithmetic estimates to geometric estimates of the mean and standard deviation from two studies of nonsmoking rural-dwelling women (Del Bubba et al., 2005; Zanieri et al., 2007). D_{oi} is the geometric average of the ith PAH taken in through dietary sources. Oral PAH Tcos for both carcinogenic and noncancer PAHs are shown in Table J.3-6.

The Italian dietary study by Lodovici et al. (1995) supplied data in which OEHHA could calculate estimates of dietary intake of nine PAHs among a population living mostly in urban settings. OEHHA obtained GM and GSD estimates by converting arithmetic estimates of dietary intake reported in Lodovici et al (1995) and estimates of intake variability from Buiatti et al (1989).

These investigators estimated that the entire study population consumes about 1.9 μg of carcinogenic PAHs per day from dietary sources. Approximately 46% of the total carcinogenic PAH intake comes from cereal products, non-barbecued meat, oils and fats. Even though meat barbecued on wood charcoal has the highest PAH levels, the contribution of these barbecued foods is only about 13% of the carcinogenic PAH intake.

A limitation of the Italian dietary intake study is that the population examined was 58% men, and did not report any body weight adjustments. Thus, the sample population may not represent the female population sampled by Zanieri et al (2007). Other studies that have compared dietary PAH intake levels between men and women indicate that men consume slightly higher levels of PAHs than women do (5% to 15% on a $\mu\text{g/kg-body weight-day}$ basis) (Falco et al 2003, Marti-Cid et al 2008), so the bias introduced by this assumption may not be significant.

Table J.3-6 presents the dietary intake and mother's milk concentrations for individual PAHs from the Italian studies. OEHHHA calculated Tcos for individual PAHs common to both the studies of dietary intake and mother's milk concentration. The mother's milk concentrations for individual PAHs represents the pooled average reported in the Zanieri et al. and Del Bubba et al. studies.

Table J.3-6: Oral Transfer Coefficients (Tcos) for Individual PAHs Based on Italian Data from a Daily PAH Dietary Intake Study (Lodovici et al., 1995; Del Bubba et al., 2005; Zanieri et al., 2007) and Mother's Milk PAH Concentration Studies (Del Bubba et al., 2005; Zanieri et al., 2007).

PAH	Mother's milk PAH concentration ($\mu\text{g/kg-milk}$)	Daily PAH intake ($\mu\text{g/d}$)	Oral PAH Tco (d/kg)
With Cancer Potency Factors	GM ^a , GSD ^b	GM, GSD	GM, GSD
Naphthalene	4.12, 1.41	NA ^c	NA
Chrysene	0.01, 3.36	0.49, 2.82	0.02, 4.93
Benzo[a]anthracene	0.12, 5.41	0.27, 2.82	0.44, 7.25
Benzo[b]fluoranthene	0.21, 3.61	0.1, 2.82	2.1, 5.21
Benzo[k]fluoranthene	0.055, 3.01	0.034, 2.82	1.62, 4.54
Benzo[a]pyrene	0.01, 3.36	0.076, 2.82	0.13, 4.93
Dibenzo[a,h]anthracene	0.007, 3.36	0.003, 2.82	2.33, 4.93
Indeno[1,2,3-c,d]pyrene	0.011, 3.36	NA	NA
Without Cancer Potency Factors	GM, GSD	GM, GSD	GM, GSD
Anthracene	0.13, 4.26	NA	NA
Acenaphthylene	4, 1.99	NA	NA
Phenanthrene	0.41, 2.03	NA	NA
Fluorene	0.12, 6.32	NA	NA
Acenaphthene	1.39, 2.16	NA	NA
Pyrene	0.15, 3.47	0.11, 2.82	1.35, 5.05
Fluoranthene	0.16, 3.34	0.6, 2.82	0.27, 4.91
Benzo[g,h,i]perylene	0.01, 3.37	0.116, 2.82	0.08, 4.94

^a GM, geometric mean; ^b GSD, geometric standard deviation; ^c NA, Not available;

Oral Tcos were calculated for each individual PAH by equation J-8. The average

Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg) and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same efficiency as some of the most common PAHs without cancer potency factors that are taken in through the diet.

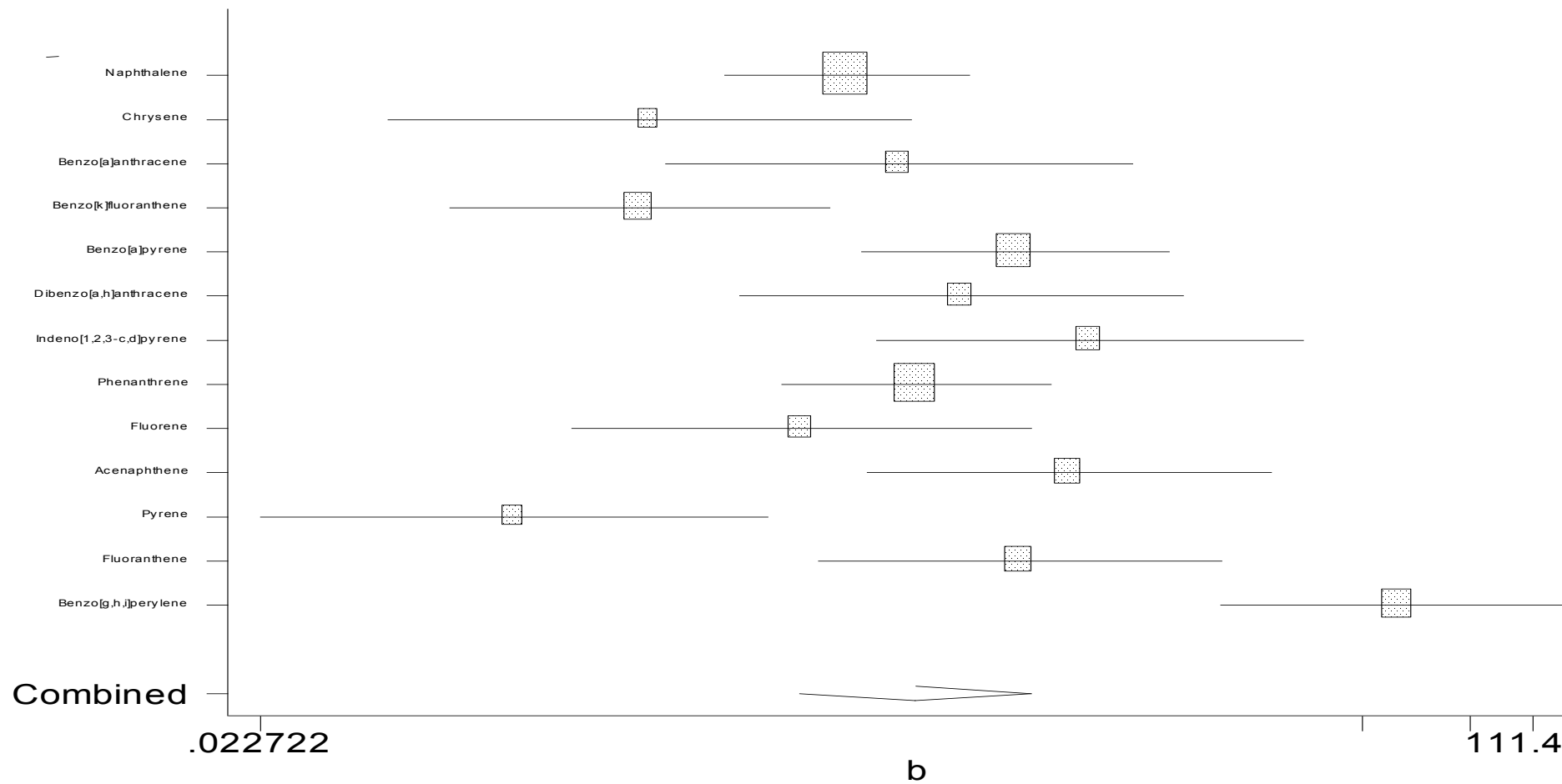
Summary Tcos were calculated using a Random-effects model to pool across individual PAH-Tcos. OEHHA found no systematic difference between summary estimates stratified by PAHs with or without cancer potency factors (data not shown). Therefore, we pooled Tcos for both groups by route of intake (see Table J.3-7).

Table J.3-7: Random Effects Estimate and 95% Confidence Intervals of Tcos Stratified by Intake Route and Data Source

Tco (data source)	No. PAHs	summary estimate (random effects model)	LCL	UCL
Inhalation	13	1.55	0.731	3.281
Oral (Italian)	9	0.401	0.132	1.218

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco.

Figure J.3-1: Inhalation Tcos (b, 95% CL) Based on Italian Data, (Random-effects Model)



Top seven estimates are PAHs with potency factors and bottom six estimates are PAHs without potency factors; summary of all 13 PAHs is labeled “combined” = 1.55 d/kg; b, the Tco in units of day/kg-milk

Similar to the inhalation Tco derivation, limitations of the oral Tco derivations include the small number of women examined for PAHs in mother's milk (n=21) and the large number of "below detection limit" results for milk concentrations, particularly for the larger PAHs with more than four rings. OEHHA assumed that the arithmetic estimates, minimum and maximum values reported by investigators represented a lognormal distribution and converted estimates from arithmetic to geometric. Nevertheless, the use of sparse data to derive an inhalation Tco and data from potentially two different study populations to generate an oral Tco – one for dietary PAH intake and another for mother's milk PAH concentrations - introduces considerable uncertainty.

J.3.3 Comparison and Use of Inhalation and Oral PAH Tcos

Comparison of the oral and inhalation Tcos also presents a number of interesting findings. For example, comparing the averaged inhalation and oral mother's milk Tcos generated from the Italian studies for carcinogenic PAHs, the mean inhalation Tco is about four times greater than the oral Tcos based on Italian study data.

Although studies in humans are lacking, (Grova et al., 2002) showed that BaP is poorly absorbed through the gut in goats when administered orally in vegetable oil. Radiolabeled BaP fed to these animals led to 88% recovery of the radioactivity in feces, indicating little BaP reached the bloodstream where it could be taken up in mother's milk. In contrast, respiratory absorption of PAHs in particulate form through smoking is about 75% efficient (Van Rooij et al., 1994).

The following factors may have influenced the difference between oral Tco values and inhalation Tco values:

- First-pass metabolism in the liver following oral intake before reaching the blood supply of the breast versus entering systemic blood circulation prior to passage through the liver with the inhalation route (however, some PAH metabolism occurs in the lung)
- Gut assimilation of PAHs is likely to occur at a different rate than the rate of passage across the lung

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of PAHs in the mother's body occurs more readily when inhaled versus ingested. Carryover rate, defined here as the daily output of PAHs in mother's milk ($\mu\text{g/day}$) over the daily intake of PAHs ($\mu\text{g/day}$), can be estimated by multiplying a PAH Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, the calculated carryover rate turns out to be the same as the PAH Tco value. A carryover rate greater than one in PAH transfer suggests that accumulation occurs in the mother's body prior to lactation.

The average inhalation Tco of 1.6 d/kg daily inhalation of a PAH mixture, indicates that 160% of the daily intake from inhaled sources transfers to mother's milk. This high transfer-value suggests that some accumulation of PAHs with cancer potency factors may occur in the mother's body before lactation begins. An average oral Tco of 0.40 d/kg for PAHs with cancer potency factors indicates 40% of the daily intake from diet transfers to mother's milk following oral intake of PAHs.

This suggests that metabolism occurs in the mother's body. The uncertainties in our Tco estimation methods could account for both of these results. If the Tco estimation is correct, the mother may be metabolizing a considerable fraction of her intake prior to partitioning into the fat stores. There could also be inefficient transfer to mother's milk for unknown reasons or metabolism following transfer of PAHs to mother's milk.

J.4 Mothers' Milk Transfer Coefficients for Inorganic Lead

Inorganic lead is naturally present on the earth's crust and may enter terrestrial and aquatic ecosystems due to the weathering of rocks. Traces of lead can not only be found in the immediate vicinity of emission sources but also are present, albeit at very low levels, in every part of the world (Castellino and Castellino, 1995).

Lead particulate matter is the primary form of lead present in the air (OEHHA 1997). Atmospheric movements may transport lead aerosol in the form of very fine particles, a long way from its place of emission. Refineries, mineral extraction industries, and smelting plants for lead and other metals are largely responsible for emitting lead-containing aerosols into the atmosphere (Castellino and Castellino, 1995) in the U.S..

Human intake of lead can occur by inhalation of airborne particles and ingestion of lead-contaminated food and water. Furthermore, people can be exposed using lead-glazed or painted cooking and eating utensils. Lead may also be ingested in foods or drinks contaminated with the metal during the industrial processes of food production or preservation (Castellino and Castellino, 1995). The potential pathways of concern with Hot Spots facilities would be inhalation, soil ingestion, and dermal absorption, home raised meat, homegrown produce, surface drinking water consumption, and breast milk consumption.

Background levels of lead in the blood of the U.S. population have declined in recent years mainly resulting from the removal of lead from gasoline and paint. Results from an NHANES study (1991 – 1994) show that the geometric mean blood lead level in the U.S. adult population (20 – 69 years of age) was about 4 µg/dL (Pirkle et al., 1994), which is over a 70% decline in blood lead from blood lead levels obtained from 1976 to 1980. The NHANES IV survey (1999- 2000)

found an additional 50% reduction (1.75 µg/dL) in the U.S. adult population (CDC, 2005).

As of the date of this report, measured levels of lead at ambient air quality monitoring sites in California are very low. Lead exposure in the California population is likely to occur from sources other than Hot Spots facility emissions, such as old lead-based paint. However, no threshold has been identified for lead-induced neurotoxicity in children and therefore an evaluation of all potential routes of exposure for Hot Spots facilities is prudent. Further, there are significant lead emissions from some Hot Spots facilities.

In an effort to derive lactation transfer coefficients for inorganic lead, OEHHA drew from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

J.4.1 Inorganic Lead in Human Milk

Breast milk levels of lead correlate with levels of lead in whole blood but are generally much lower (Sternowsky and Wessolowski, 1985; Castellino and Castellino, 1995; Li et al., 2000; Ettinger et al., 2004). Castellino et al (1995) reviewed 11 studies conducted between 1933 to 1989 and observed that in the vast majority of cases, the mean values of lead in breast milk vary from 0.17 to 5.6 µg/L (Castellino and Castellino, 1995).

Ursinyova and Masamova (2005) published a table of 32 human milk summary estimates from studies published between 1983 and 2001. Mean human milk levels of lead generally ranged from 0.5 to 50 µg/L (Ursinyova and Masanova, 2005). Average blood lead levels during that timeframe ranged from 24 to 460 (µg/L) (Gulson et al., 1998a).

Because lead levels in milk correlate well with whole blood, OEHHA searched for studies that reported both lead levels in milk and blood before and/or during lactation for derivation of a lactational Tco for lead. However, several investigators have questioned high results from early studies of lead in breast milk. For example, Ettinger et al (2004), Gulson (1998b) and others cautioned that high levels of lead in breast milk might be due to contamination from some past sample collection techniques (Hu et al., 1996; Newman, 1997; Gulson et al., 1998a; Smith et al., 1998; Ettinger et al., 2004). These sources of lead include the use of the following products to prepare nipples or express breast milk:

- lead acetate ointment
- lead in nipple shields
- lead in alcohol wipes from foil wrap

Gulson et al (1998a) also suggested that analytical problems, indicated by an unusually wide range in lead concentrations for the quality control standard in Parr et al (1991), warrant verification by follow-up studies (Parr et al., 1991; Gulson et al., 1998a). Gulson et al (1998a) assessed lead concentrations in maternal blood versus the concentration of lead in breast milk per concentration in maternal whole blood from studies conducted over 15 years prior to 1998.

From this assessment, they suggested that milk lead levels less than about 15% of maternal blood lead levels best represent the relationship between lead in maternal blood and milk. In other words, milk lead levels that were greater than 15% of blood lead levels were suspected of being contaminated with lead during sample collection and/or assessment. Therefore, OEHHA has included only summary estimates from studies published after 1990 that did not report or show evidence of breast milk contamination.

OEHHA located eight studies that met our inclusion criteria. Table J.4-1 summarizes key attributes of the study populations.

Table J.4-1: Studies with Summary Estimates of Concurrent Maternal Blood and Milk Levels of Lead)

Study	Country	Group	Study period	Measurement	# Study subjects
(Nashashibi et al., 1999)	Greece	Residents of Athens and surrounding areas	~1999	At delivery, at onset of lactation	47
(Li et al., 2000)	China, Shanghai	Not occupationally exposed	prior to 2000	At delivery, at onset of lactation	32
(Counter et al., 2004)	Ecuador, Pujili	Pottery glazers	2003	Post partum	13
(Ettinger et al., 2004)	Mexico, Mexico City	Exclusive breast feeders	1994-1995	One month postpartum	88
(Ettinger et al., 2004)	Mexico, Mexico City	Partial breast feeders	1994-1995	One month postpartum	165
(Namihira et al., 1993)	Mexico (Mexico City)	Reside near New Smelter	1986	postpartum	35
(Hallen et al., 1995)	Sweden	Reside in Rural areas	1990-1992	6 weeks postpartum	39
(Hallen et al., 1995)	Sweden	Reside near Smelter area	1990-1992	6 weeks postpartum	35
(Baum and Shannon, 1996)	U.S.A Camden, New Jersey	Mothers of lead poisoned infants	1996	Postpartum	2
(Gulson et al., 1998b)	Australia	Immigrants from eastern Europe	Early 1990s	At delivery and average during lactation	9

Regression analyses suggest a linear relationship between lead in maternal blood and milk among women with substantially elevated levels of lead in blood. For example, Namihira et al (1993) reported a significant linear relationship ($r = 0.88$) between levels of lead in blood and milk for blood lead levels in the range of 35 µg/dL -100 µg/dL from a study of 35 lactating women living in Mexico City (Namihira et al., 1993). At these levels of lead in blood, authors reported a univariate regression of 4.3% representing the average level of lead in breast milk relative to the average level of lead in blood.

A similar study of 47 lactating women conducted by Nashashibi et al also reported a significant linear relationship ($r=0.77$) between lead in milk and blood for blood lead levels in the range of 5 µg/dL - 25 µg/dL (Nashashibi et al., 1999).

Based on a univariate regression, the average level of lead in breast milk was about 7% the average level of lead in blood. OEHHA calculated similar estimates of the milk/blood lead ratio from Li et al (2000), Counter et al (2002) and Ettinger et al (2004) (see Table J.4-2).

Table J.4-2 Concurrent Measurements of the Lead Concentration ($\mu\text{g/L}$) in Mother's Milk and Blood

Study		Blood	Milk	Blood	Milk
	N	AM,SD	AM,SD	GM,GSD	GM,GSD
(Nashashibi et al., 1999)	47	149, 41.1	20,5	143.64, 1.31	19.4, 1.28
(Li et al., 2000)	119	142.5, 69.14	5.63,4.39	128.21, 1.58	4.44, 1.99
(Counter et al., 2004)	13	171, 91	4.6,5.3	150.96, 1.65	3.02, 2.51
(Ettinger et al., 2004)	88 ^a	94, 48	1.4,1.1	83.72, 1.62	1.1, 2
(Ettinger et al., 2004)	165 ^b	95, 43	1.5,1.2	86.55, 1.54	1.17, 2.02
(Namihira et al., 1993)	35	459, 198.8	29.94,25.75	421.19, 1.51	24.7, 1.86
(Hallen et al., 1995)	39 ^c	31.4, 6.7	0.5,0.3**	30.71, 1.23	0.43, 1.74
(Hallen et al., 1995)	35 ^d	31.7, 10.2	0.9,0.4***	30.18, 1.37	0.82, 1.53
(Baum and Shannon, 1996)	2	315, 35.4	5.02,0.50	313.03, 1.12	5, 1.1
(Gulson et al., 1998b)	9	29, 8	0.73,0.7	27.96, 1.31	0.53, 2.24

^aexclusively breast fed; ^b partially breast fed; ^c rural setting; ^d near smelter; * < LOD taken as 1/2 LOD as GM and 9.9 = max, **based on LOD of 0.5 $\mu\text{g/L}$ and 2 out of 39 samples above LOD; *** based on 16/35 above LOD

Li et al (2000) stratified milk lead levels by low, medium and high blood lead levels. Their findings suggest that slightly higher transfer rates occur at low levels relative to high levels of lead in blood (Li et al., 2000). This may be due to more efficient transfer rates at lower body burdens of lead or it could result from very slight breast milk contamination during collection and/or assessment.

J.4.2 Biotransfer from Bone to Blood during Pregnancy and Lactation

Lead transferred from blood to human milk reflects both the mother's current and ongoing intake of lead exposure as well as lead mobilized due to physiological changes of pregnancy and lactation from bone stores due to past exposures. Several studies provided indications of internal transfer of lead from bone stores. Internal transfer was evident by comparing the rise in blood lead levels during lactation to blood lead levels measured prior to lactation (see Table J.4-3).

Table J.4-3: Change in Blood Lead Levels from Pregnancy (bloodpreg) to Lactation (bloodlac) ($\mu\text{g/L}$)

Study	N	Bloodpreg AM,SD	Bloodlac AM,SD	Bloodpreg GM,GSD	Bloodlac GM,GSD
(Gulson et al., 1997)**	8	22.4, 6	32, 8.4	21.64, 1.30	30.95, 1.29
(Ettinger et al., 2004)	~86-88excl	81, 38	94, 48	73.33, 1.56	83.72, 1.62
(Ettinger et al., 2004)	164-165part	90, 44	95, 43	80.85, 1.59	86.55, 1.54
(Tellez-Rojo et al., 2002)	425	84, 40	93.7, 43.04	75.84, 1.57	85.15, 1.55
(Sowers et al., 2002)*	15	13.7, 7.75	17, 5.29	11.93, 1.69	16.23, 1.36
(Rothenberg et al., 2000)	311	27.59, 26.49	32.03, 21.78	22, 1.96	28, 1.68

* SD for blood lead level during lactation estimated for blood lead at 6-months from figure 2; ** bloodlac is max blood lead level during pregnancy and lactation; excl, exclusively breastfed; part, partially breastfed

These investigators conducted longitudinal monitoring of blood samples to determine stable lead isotope profiles by mass spectrometry and chemical analyses of blood samples for total lead content over a 300-day period. Gulson et al followed Australian women (15 immigrants and 7 non-immigrants) to study the mobilization of lead from the maternal skeleton during pregnancy and lactation (Gulson et al., 1995; Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Gulson et al., 1999; Gulson et al., 2001). Investigators measured maternal and infant blood, urine, diet, and breast milk from 21 mothers and 24 infants. The arithmetic mean and standard deviation lead concentration in breast milk were AM (SD) 0.73 (0.70) $\mu\text{g/kg}$ and the geometric mean and standard deviation were GM (GSD) 0.55 (2.24) respectively. Levels ranged from 0.09 to 3.1 $\mu\text{g/kg}$.

Gulson et al (1997) provided evidence that lead in female immigrants to Australia was mobilized from skeletal stores during pregnancy, with increases in blood lead concentration of about 20% and a mean increase in skeletal lead contribution to blood lead of 31%. Authors concluded that between 45% and 70% of lead in blood comes from mobilized long-term tissue lead stores (Gulson et al., 1997).

Investigators obtained environmental samples of house dust, drinking water, urban air, gasoline, and a 6-day duplicate diet quarterly. The GM (GSD) blood lead concentration for the immigrant females on arrival in Australia (either prior to or during early pregnancy) was 3.0 $\mu\text{g/dL}$ (1.56) (range: 1.9 to 20 $\mu\text{g/dL}$) and for the Australian controls was 3.1 $\mu\text{g/dL}$ (range: 1.9 to 4.3 $\mu\text{g/dL}$). Skeletal lead

contribution to blood lead was significantly greater ($p < 0.001$) during the post pregnancy period than during the 2nd and 3rd trimesters.

The contribution of skeletal lead to blood lead during the post-pregnancy period remained constant at the increased level even though the duration of breast-feeding varied from 1 week to 6 months. The Authors concluded that the increased contribution of skeletal lead both during pregnancy and in the post pregnancy period is consistent with increased bone resorption and may be associated with inadequate calcium intake.

Sowers et al (2000) followed lactating women enrolled in prenatal program located in Camden, New Jersey between 1997 and 2000 (Sowers et al., 2002). These women were part of a larger cohort of 962 women enrolled in study of calcium metabolism in pregnancy and lactation. A nested cohort of 15 women with a mean (standard deviation) age of 23.7 (5.42) years, who provided breast milk samples through 6 months postpartum or longer and were unaware of their blood lead levels, was included in the study. Blood and milk lead levels along with measures of bone loss and osteocalcin concentrations were evaluated. Authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

The arithmetic mean (standard deviation) ($\mu\text{g/dL}$) of blood lead levels at delivery for 15 breast-feeding and 30 randomly selected bottle-feeding women were 1.37, (0.78), and 1.31, (1.10) respectively. Mean maternal blood lead levels rose to 1.6, (1.7) $\mu\text{g/dL}$ at three and six months during lactation, respectively. Compared to bottle-feeding women, blood lead levels from breast-feeding women were consistently higher by 15 – 35% during the first six months postpartum. Authors found that breast-feeding women had greater bone loss as reflected in the bone change data and higher serum osteocalcin concentrations than bottle-feeding women.

The arithmetic mean of lead in breast milk samples (standard deviation) were 5.6 (4.2) and 5.9 (3.87) $\mu\text{g/L}$ at three and six months post partum. Breast milk lead was also measured 1.5 and 12 months post partum. However, authors did not measure blood lead at 1.5 months, did not indicate how many women were still breast-feeding nor did they attempt to estimate how many liters/day study subjects produced. The relative increase in blood lead levels from delivery to an active lactating period (e.g. one to 6 months) is consistent with the relative increases in blood lead found in other studies (see Table J.4-3).

Tellez-Rojo et al (2002) concluded that maternal bone lead levels are an important predictor of maternal blood lead levels over the course of lactation. In fact, bone lead from past exposures can contribute an additional 40% of the lead measured in blood during lactation (see Table J.4-3) (Tellez-Rojo et al., 2002).

Ettinger et al (2004) measured relatively high maternal blood lead levels in women exposed to lead in the air while living in Mexico City. Between January 1994 and June 1995, investigators selected 1398 women from three maternity hospitals in Mexico City for participation in a randomized control trial (Tellez-Rojo et al., 2002; Hernandez-Avila et al., 2003; Ettinger et al., 2004). From this study population, 629 women agreed to participate. Ettinger et al., (2004) examined a nested cohort of 255 women with a mean (standard deviation) age of 24 (5) years with both breast milk, maternal and infant blood lead levels at delivery and one-month post partum. The Authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

For breast-feeding women, the arithmetic mean (standard deviation) of blood lead level at delivery was 8.7 (4.2) and at one-month post partum was 9.4 (4.5) $\mu\text{g/dL}$. At one-month post partum, the average (standard deviation) lead level in breast milk was 1.5 (1.2) $\mu\text{g/L}$. After adjusting for parity, calcium intake, infant weight change and breastfeeding status, an increase in blood lead was associated with a 33% increase in breast milk lead.

Rothenberg et al (2000) recruited immigrant women, almost exclusively from Latin America, from outpatient clinics in South Central Los Angeles to examine bone lead contribution to blood lead. Investigators contacted subjects from June 1995 through July 1998. Three hundred eleven subjects were followed from late pregnancy to one or two months after delivery. The Investigators evaluated bone lead levels after delivery and blood lead levels both pre- and post-delivery. Ages ranged from 15 to 44 years. Prenatal blood lead was lower on average $\text{GM} = 2.2 \mu\text{g/dL}$ (0.4 to 38.7) than postnatal blood lead $\text{GM} = 2.8 \mu\text{g/dL}$ (0.4 to 25.4). In fact, post natal blood lead level increased by 27% relative to the prenatal blood lead level.

A questionnaire was administered including questions about present breast feeding practice (presently nursing yes/no) and past history of breast feeding (ever nursed and total months nursed). Breast milk samples were not obtained from this cohort. Tibia and calcaneus bone lead levels were associated with prenatal blood lead levels and calcaneus but not tibia lead was associated with postnatal blood lead levels (Rothenberg et al., 2000).

J.4.3 Inhalation Biotransfer of Lead to Mother's Milk

Ideally, lead transfer to human milk would include estimates of lead in ambient air and major sources of oral exposure over time along with human milk estimates from the exposed lactating population. However, few studies have attempted to correlate lead exposure from multiple pathways (e.g. oral sources such as contaminated food, water, dust and soil and inhalation sources such as ambient air) with lead concentrations in human mother's milk. This is likely due to the multiple effects of daily intake from environmental sources (Sannolo et al., 1995) and internal transfer from lead released from bone stores during pregnancy and

lactation (Gulson et al., 1997).

Although exposure to lead can come from many sources, ambient air contaminated from combustion sources has been a significant source of exposure in the U.S. population and European countries (U.S. EPA 1998). The relationship between air lead and blood lead has been studied extensively in both field studies and experimental chamber studies. OEHHA evaluated studies conducted prior to 1997 in their health risk assessment of inorganic lead under the toxic air contaminant program (OEHHA, 1997).

Briefly, in the OEHHA report, the contribution of airborne lead to blood lead levels was examined using several different methods – disaggregate, aggregate, uptake biokinetic, and physiologically based pharmacokinetic models (OEHHA, 1997). Findings were evaluated for linearity over a wide range of air and blood lead levels and are expected to apply to some exposure scenarios under the Hot Spots program. Most of these studies were conducted prior to 1985 when both air and blood lead levels were much higher than they are now. For example, the level of lead in the air used in chamber studies was $3.2 \mu\text{g}/\text{m}^3$ representing low exposure and $10.9 \mu\text{g}/\text{m}^3$ representing high exposure, while background air was typically between $7 \mu\text{g}/\text{m}^3$ and $8 \mu\text{g}/\text{m}^3$ in the city of Los Angeles during similar time-periods – late 1960s / early 1970s. Lead in Los Angeles air is 100-fold lower today (Ospital et al., 2008).

The relationship between air lead concentration and blood lead is not linear. Higher slopes are observed at lower air lead concentrations. However, the aggregate model was chosen because it implicitly incorporates all air-related pathways (i.e. soil, dust, water, contaminated food, etc.) and has averaged slopes estimated from a wide range of air concentrations. Using this model OEHHA estimated that an average change in adult blood lead ($\mu\text{g}/\text{m}^3$) per lead in air concentration (m^3/dL) of 1.8 for ambient air levels in California.

As part of our effort to estimate a lactational transfer factor for lead (T_{co}), we searched for studies that examined slope factors in other populations or were conducted subsequent to our 1997 report (OEHHA, 1997).

In addition to the kinetics of lead in the general adult population, recent studies have observed that - under similar exposure conditions - plasma lead rises by about 20% – 80% during lactation (Gulson et al., 1997; Gulson et al., 1998b; Gulson et al., 1999; Rothenberg et al., 2000; Tellez-Rojo et al., 2002). Findings from these and other investigations suggest that, in addition to daily environmental sources of exposure, breast milk levels of lead also reflect lead released from lead accumulated in the lactating woman's bones.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated

estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

J.4.4 Population Transfer Coefficient (Tco) for Lead

OEHHA has derived transfer coefficients for lead using Equation J-9

$$Tco_{hma} = (C_{ma}/C_{blood}^{+}) \times (C_{blood}^{+}/C_{blood}) \times (C_{blood}/(C_{air} \times BR)) \times F_{c1} \times F_{c2} \quad \text{Eq. J-9}$$

where:

C_{ma} = geometric mean human milk lead level ($\mu\text{g/L}$ -milk as wet weight)
 C_{blood}^{+} = geometric mean blood lead level during lactation ($\mu\text{g/dL}$)
 C_{blood} = geometric mean blood lead level during non-lactating state ($\mu\text{g/dL}$)
 C_{air} = geometric mean concentration of lead in ambient air ($\mu\text{g/m}^3$)
 BR = geometric mean breathing rate for adult women ($14 \text{ m}^3/\text{day}$)
 F_{c1} = conversion factor ($\text{L-milk}/(\text{kg-milk}) \sim (0.97)$)
 F_{c2} = conversion factor ($\text{dL}/(\text{L}) = 10$)

C_{ma} is the geometric mean human milk lead level that incorporates all (aggregated) air-related pathways of lead. C_{blood}^{+} is the geometric mean blood lead level among lactating women measured during lactation ($\mu\text{g/L}$). C_{blood} is the geometric mean blood lead level taken from the general population during a non-lactating state ($\mu\text{g/L}$). C_{air} is the geometric mean concentration of lead in the ambient air ($\mu\text{g/m}^3$) inhaled by the same population where blood lead levels were measured. BR is the geometric mean breathing rate for adult women ($14 \text{ m}^3/\text{day}$) (see Chapter 2). F_{c1} is the inverse of the specific gravity of breast milk (1.03 g/ml) (Sergen, 2006). F_{c2} is the conversion from deciliters to liters.

J.4.4.1 Biotransfer from Blood to Milk

Three groups measured maternal blood lead before and during lactation along with lead in mother's milk (Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Sowers et al., 2002; Ettinger et al., 2004). However, Sowers et al. reported unusually high levels of lead in breast milk relative to blood, which suggest contamination problems. It is possible that breast milk samples were contaminated by the sampling collection technique (e.g. lead in the nipple shields). However, it is also possible that a more efficient active transport mechanism at lower blood lead levels could explain higher levels of lead in breast milk relative to blood. More studies of mothers with low blood lead levels are needed to further verify the results reported by Sowers et al.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels before the onset of lactation, during lactation and relative to the levels of lead in breast milk

(Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Ettinger et al., 2004).

J.4.4.2 Transfer from Air to Blood

Equation J-10 describes estimation of aggregate transfer from airborne and associated sources that appears in the OEHHA 1997 report on the health effects of airborne inorganic lead (OEHHA, 1997):

$$\text{Slope factor} = (C_{\text{blood e}} - C_{\text{blood r}}) / (C_{\text{air e}} - C_{\text{air r}}) \quad \text{Eq.-J-10}$$

$(C_{\text{blood e}} - C_{\text{blood r}})$ is the difference between lead concentration in the blood of exposed compared to reference group and $(C_{\text{air e}} - C_{\text{air r}})$ is the difference in air lead between exposed and reference group. This simplified model assumes that the exposed and reference communities are similar in confounders such as age and smoking habits and reasonably comparable in their exposure to other sources of lead (e.g. paint).

Subsequent to OEHHA's 1997 report, Ranft et al (2008) published results from studies conducted on exposure to air pollutants among residents living near industrial sources along the rivers Rhine, Ruhr and Wupper in North Rhine-Westphalia Germany during five time-periods from 1983 to 2000. Authors reported the distribution of ambient air lead levels for each of the five time-periods (Ranft et al., 2008).

During the early years (1983 – 1991), ambient air lead levels ranged from 0.100 – 0.510 $\mu\text{g}/\text{m}^3$. Whereas, during the later years (1997 – 2000), air lead levels were much more variable - ranging from 0.025 to 0.729 $\mu\text{g}/\text{m}^3$. The 50th percentile (P 50) declined by almost a factor of 20 from years 1983 to 2000. During the earliest years (1983 – 1991), P 50 declined by a factor of four from 0.465 to 0.100 $\mu\text{g}/\text{m}^3$. Based on data collected from 1991 to 2000, these investigators reported that childhood blood lead would decrease by a factor of 6.4: 95%CI (6.02 – 6.80) from the decrease in lead concentration in polluted ambient air (m^3/dL).

OEHHA calculated a similar slope factor from the study of 500, 55-yr-old women living in industrial areas of the North Rhine – Westphalia, Germany from 1985 to 1990 by Wilhelm and associates (Wilhelm et al., 2007). These investigators reported that mean blood lead levels among these women declined from 7.2 to 5.0 $\mu\text{g}/\text{dL}$. Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead ($\mu\text{g}/\text{dL}$) over a similar period (GM, 6.2; 95% CL 6.1 – 6.4)(Ranft et al., 2008). This estimate is within the range of slope factors reported previously by OEHHA for the general adult population (OEHHA, 1997).

J.4.3.3 Transfer from Air and Body Stores to Milk

Tables J.4-4 and J.4-5 show the Tcos derived by combining air to blood and blood to milk transfer of inorganic lead from the available data. Table J.4-4 shows the transfer factors derived from the study of eight women who provided samples of blood before and during lactation as well as samples of milk during lactation (Gulson et al., 1998a; Gulson et al., 1998b). The geometric mean and standard deviation blood lead levels prior to lactation were low (GM 2.2 µg/dL, GSD1.3).

Table J.4-4: Transfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Gulson et al., 1998a; Gulson et al., 1998b) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m ³ /dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.024	3.19	0.009	0.061
Willhelm/Ranft	6.2	0.08	3.19	0.031	0.203

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.4-5 shows the transfer factors derived from the study of 253 women who provided samples of blood prior-to and during lactation as well as samples of milk during lactation (Ettinger et al., 2004).

Table J.4-5: Biotransfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Ettinger et al., 2004) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m ³ /dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.019	3.00	0.017	0.022
Willhelm/Ranft	6.2	0.064	3.00	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Compared to Gulson et al (1998), the geometric mean, blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively)(Gulson et al., 1998b; Ettinger et al., 2004).

However, the transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two.

J.4.4 Study Limitations, Influencing Factors and Uncertainty (inorganic compounds)

Our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5 Summary and Recommendations

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHHA derived human lactational transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral exposure (e.g. air, cigarette smoke or diet) in the same or a similar human population. These coefficients can be applied to the mother's chronic daily dose estimated by the Hot Spots exposure model to estimate a chemical concentration in her milk.

We established transfer coefficients (Tcos) for individual congeners and WHO-TEQ summary PCDDs/Fs and dioxin-like-PCBs, individual and summary carcinogenic PAHs, and lead through equations J-1-3, data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination is constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate over the lactation period. In some cases, OEHHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHHA describes the method of adjustment in the text and table containing adjusted values.

We described the methods for deriving specific Tcos from measurements of human milk, intake and transfer estimates from studies of populations exposed to general global sources of pollutants. Although the proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from exposure found with global sources, we believe Tcos in this appendix have been derived from data that serve as reasonable surrogates of transfer from Hot Spot facility exposures.

J.5.1 Dioxins and Furans

Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Milbrath et al 2009, Flesch-Janys et al 1996). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce

the maternal body burden of PCBs during 6 months of lactation (Niessen et al 1984, Landrigan et al. 2002).

Therefore, OEHHA incorporated conservative assumptions regarding these factors into our model (i.e. reference half-lives based on body burden below 700 ppt in the blood, adult age, nonsmoker, no recent prior breast-feeding period and percent body fat of older adults) in addition to accounting for the substantial variability between individual congeners of PCDDs, PCDFs and dioxin-like PCBs.

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in the adult human body derived from dietary and occupational exposures. OEHHA estimated oral Tcos for these chemicals from estimates of body weight reported in Chapter 10 of this document, the steady-state equation developed by Smith (1987) and reference half-lives reported in Milbrath et al (2009). Milbrath et al (2009) adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. An average dioxin Tco of 3.7 d/kg indicates that 370% of the daily intake from ingested sources transfers to mother's milk. This high transfer-value suggests that some accumulation of carcinogenic dioxins and dioxin-like compounds occurs in the mother's body. For individual congeners, an oral Tco less than one (e.g. 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggests that some metabolism occurs in the mother's body.

J.5.2 PAHs

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers (Zanieri et al. 2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1.

The key assumption underlying the development of these Tcos is that the variability in individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

OEHHA calculated oral Tcos for each individual PAH by Equation J-8. The average Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same efficiency as some of the most common PAHs without cancer potency

factors that are taken in through the diet. Therefore, summary Tcos were calculated by pooling across individual PAH-Tcos from both groups (see Table J.3-7).

J.5.3 Inorganic Lead

In an effort to derive lactational transfer coefficients for inorganic lead, OEHHA has drawn from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels due to the onset of lactation as well as during lactation relative to the levels of lead in breast milk.

Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead ($\mu\text{g/dL}$) over a similar period (GM, 6.2; 95% CL 6.1 – 6.4).

Compared to Gulson et al (1998), the geometric mean blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively) (Gulson et al., 1998b; Ettinger et al., 2004).

The transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two. However, our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5.4 Recommendations

OEHHA recommends using the Tcos based on the summary estimates provided in Table J.1-1 rather than the individual compound Tcos provided in Tables J.2-3, J.3-4, and J.3-6 to assess transfer of compounds to mother's milk. Tcos of individual compound are less robust than summary Tcos listed in Table J.1-1 because in some cases they have derived from data containing a high number of non-detects and small sample sizes. Additional studies might improve the

estimation of individual Tco values, especially studies that incorporate more sensitive methods for analyzing breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence interval of the mean Tco; UCL, upper 95% confidence interval of the mean Tco

When calculating cancer risk from speciated PCDD/Fs, dioxin-like PCBs and PAHs, assume that the ratios of congeners measured in the emissions are preserved when transferred from the mother's body to breast milk. OEHHA recommends a single Tco for each chemical group (e.g. PCDDs oral). Risk assessors can apply TEQs to the infant dose after applying the Tco for a chemical group to each congener in the group to calculate infant cancer risk for the mother's milk pathway.

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Appendix K
Meat, Milk, and Egg Transfer Coefficients

K.1 Chemical Transfer Coefficient (Tco) Derivation Methodology

Meat, cow's milk and eggs can become contaminated when food-producing animals inhale or ingest contaminated materials that then transfer into these food products. The transfer coefficients (Tco) presented in Tables K.1 and K.2 were derived from published studies investigating chemical concentrations in food products resulting from animal intake of the chemical. In most studies, the chemicals were mixed into the animal's feed, although some studies investigated the bioaccumulation of chemicals from contaminated soil in poultry feed. The Tcos, expressed in day/kilogram (d/kg), represent the ratio of contaminant concentration in fresh weight animal product (in mg/kg, for example) to the daily intake of contaminant by the animal (in mg/day). Tcos were determined only for the main food-producing animal sources, including cow's milk, eggs, and meat from cattle, pigs and chickens.

The studies selected to estimate Tcos were usually of long enough duration to allow steady-state concentrations to be reached in milk-, meat- and egg-producing animals. Steady-state concentrations in the tissues are a function of the tissue elimination half-lives (MacLachlan and Bhula, 2008). Assuming a first-order process, an exposure duration that is five times greater than the tissue elimination half-life has been used to represent time to steady-state conditions (i.e., the ratio of the measured concentration at five half-lives to steady-state concentration is 0.968).

Realistically, fast-growing animals used for food may never attain a true tissue steady-state for persistent organic chemicals due to the competing factors of growth, fattening and lactation (Fries, 1996; Hoogenboom, 2005). A steady-state concentration in food-producing animals will likely be reached more quickly than in humans due to these factors and may even show declining levels in fat during the fattening phase of the animals prior to slaughter (Fries, 1996). The most practical approach is to base the Tco on exposure studies that expose the animal for a majority of the animals' life span up to or near marketable weight. The studies that followed tissue and milk contaminant levels during exposures over most of the animals' productive lifespan have shown that a sufficient semblance of steady-state is reached during the productive life of lactating dairy cattle and laying hens, and in meat animals prior to slaughter.

Default consumption rates of contaminated feed were used for estimating Tcos if no consumption data was provided in the primary studies. Usually, the food-producing animals in biotransfer studies were caged or treated similar to commercial farming practices. However, this exposure assessment document is primarily concerned with small farm or family farm situations in which the food-producing animals may be allowed to roam more freely than in commercial operations. This is particularly relevant for pigs and chickens. Free-range and organic farming will result in greater feed intake, slower growth, and potentially

greater contaminant exposure from range forage and soil ingestion (MacLachlan, 2010).

Specifically regarding poultry food products, the term “poultry” refers to a number of avian species that are food sources for humans. Due to the substantial human consumption of eggs and meat from chickens, the Tcos described here were exclusively based on data from chickens, laying hens (usually Leghorns) for the egg Tcos and usually meat chickens (broilers) for the meat Tcos. However, these values could also be reasonably applied to other home-raised avian species, such as turkeys and quail.

Compared to chickens and dairy cattle, fewer swine and beef cattle exposure studies could be found to estimate the biotransfer of ingested contaminants to muscle tissue. Rather than simply adopting the same cattle Tco values for swine when biotransfer data is lacking, contaminate transfer models are employed by OEHHHA to estimate differences in chemical accumulation among livestock. For transfer of organic lipophilic chemicals, MacLachlan (2009) developed Physiologically Based Pharmacokinetic (PBPK) models to derive scaling factors that are used to assist the extrapolation of transfer studies, carried out most often on lactating dairy cows, to beef cattle and pigs. Given the estimated half-life (or extraction ratio for liver) of the chemical in the animal and the ratio of the chemical concentration in milk fat to body fat of dairy cows, the appropriate scaling factor can be selected and combined with the Tco derived from lactating dairy cattle to improve estimates of residues in beef cattle and pigs.

For metal Tcos, a metabolic weight adjustment can be made that accounts for differences in tissue transfer of chemicals in animals of different weight (i.e., a lower metabolic rate is expected in larger animals such as cattle compared to smaller animals such as swine, resulting in slower rates of transfer into tissues). A similar metabolic weight approach has been used to estimate the transfer of metals to dairy cattle from data in sheep (Crout *et al.*, 2004). This adjustment is reasonable considering most of the metal compounds of interest have passive uptake and elimination processes and are subject to little or no metabolism.

The effect of metabolic weight is apparent when comparing the meat Tco values between chicken and cattle in Tables K-1 and K-2. Where published data were used to directly estimate individual chemical Tco values, the chicken Tcos were greater than cattle Tcos. For chemicals in which biotransfer could not be estimated from published reports in pigs, a default meat Tco was estimated with the following formula:

$$\text{Pig Tco}_i = (W_{\text{cow}}^{0.75} / W_{\text{pig}}^{0.75}) \times \text{cow Tco}_i \quad \text{Eq. K-1}$$

Where:

- $W_{\text{cow}}^{0.75}$ = live-weight in kg of a cow to the 0.75 power
- $W_{\text{pig}}^{0.75}$ = live weight in kg of a pig to the 0.75 power
- Pig Tco_i = pig meat Tco for chemical i
- Cattle Tco_i = cow meat Tco for chemical i

Using average live weights of 500 kg for cattle and 60 kg for swine, the metabolic weight ratio adjustment is 4.8.

Table K.1 Food Animal Transfer Coefficients for Persistent Organic Chemicals

Organic Chemical	Tcos (d/kg) ^a				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Diethylhexylphthalate	9×10^{-5}	0.04	0.002	6×10^{-4}	5×10^{-4}
Hexachlorobenzene	0.02	20	10	0.2	0.08
Hexachlorocyclohexanes	0.01	7	5	0.2	0.09
PAH's	0.01	0.003	0.003	0.07	0.06
PCB Congeners					
77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2.	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated	0.01	10	7	0.2	0.5
Unspeciated (TEQ) ^b	0.01	20	10	0.7	0.3
PCDD/F's Congeners					
2378-TCDD	0.02	10	9	0.7	0.1
12378-PeCDD	0.01	10	9	0.3	0.09
123478-HxCDD	0.009	10	6	0.3	0.2
123678-HxCDD	0.01	10	6	0.4	0.1
123789-HxCDD	0.007	7	3	0.06	0.02
1234678-HpCDD	0.001	5	2	0.05	0.2
OCDD	0.0006	3	1	0.02	0.1
2378-TCDF	0.004	10	6	0.1	0.02
12378-PeCDF	0.004	30	10	0.1	0.01
23478-PeCDF	0.02	10	8	0.7	0.09
123478-HxCDF	0.009	10	5	0.3	0.1
123678-HxCDF	0.009	10	6	0.3	0.09
234678-HxCDF	0.008	5	3	0.3	0.06
123789-HxCDF	0.009	3	3	0.3	0.03
1234678-HpCDF	0.002	3	1	0.07	0.06
1234789-HpCDF	0.003	3	1	0.1	0.02
OCDF	0.002	1	0.6	0.02	0.03
Unspeciated	0.001	6	5	0.03	0.09
Unspeciated (TEQ) ^b	0.005	10	7	0.2	0.09

^a All Tco values were rounded to the nearest whole number^b TEQ-adjusted Tco values

Table K.2 Food Animal Transfer Coefficients for Inorganic and Organic Metals

Inorganic and Organic Metals	Tcos (d/kg) ^a				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Arsenic	5×10^{-5}	0.07	0.03	2×10^{-3}	0.01^b
Beryllium	9×10^{-7}	0.09	0.2	3×10^{-4}	0.001
Cadmium	5×10^{-6}	0.01	0.5	2×10^{-4}	0.005
Chromium (VI)	9×10^{-6}	NA ^c	NA	NA	NA
Fluoride	3×10^{-4}	0.008	0.03	8×10^{-4}	0.004^b
Lead	6×10^{-5}	0.04	0.4	3×10^{-4}	0.001^b
Mercury					
Hg(II) only in diet:					
Inorganic mercury	7×10^{-5}	0.3	0.02	4×10^{-4}	0.002^b
Methylmercury (MeHg)	NA	0.5	0.09	NA	NA
MeHg only in diet:					
Inorganic mercury	NA	NA	NA	NA	NA
Methylmercury	7×10^{-4}	10	10	NA	NA
Nickel	3×10^{-5}	0.02	0.02	3×10^{-4}	0.001
Selenium	0.009	3	0.9	0.04	0.5

^a All Tco values were rounded to the nearest whole number.

^b The meat Tco was estimated using the metabolic weight adjustment ratio of 4.8 from cattle to pig

^c NA – no data available or was not applicable

Speciated data existed that allowed the derivation of individual TCos for polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-p-dioxins and furans (PCDD/F), shown in Table K.1, that are a toxicological concern under the “Hot Spots” program. TCos for unspeciated mixtures of PCBs and PCDD/Fs have also been calculated. The unspeciated TCos are the values used to estimate the impact of these two groups of compounds in risk assessments, unless chemical analysis was conducted to speciate these compounds. In addition, toxic equivalent (TEQ)-adjusted TCos have been estimated for unspeciated mixtures, which adjusts for the different toxic potencies of the congeners compared to a sentinel compound (TCDD for both PCDD/Fs and PCBs). Many of the TEQs originated from European studies and are referred to as WHO-TEQs or International-TEQs (I-TEQs). However, some more recent revisions of individual TEQ values have been carried out and are discussed in detail in OEHHA’s Air Toxics Guidelines (OEHHA, 2005). Nevertheless, the TEQ-adjusted TCos presented here probably would not vary greatly compared to recent OEHHA TEQ estimates, and provide a reasonable optional screening estimate of the overall potency of PCB and PCDD/F mixtures that are found in contaminated food products.

K.2 Tco Derivations for Milk, Meat and Eggs

K.2.1 Semi- and Non-Volatile Organic Chemicals

The exposure studies used to derive organic compound Tcos often normalized the muscle tissue, egg and cow's milk contaminant concentrations to their respective fat content. The Tcos presented here are based on fresh, whole meat, egg and milk concentrations of the contaminants. If necessary, the fat concentration of a chemical was adjusted to the average fresh weight concentration using fat content default factors derived from reference sources: 0.11 for egg, 0.07 for chicken meat, 0.19 for beef cattle meat, 0.23 for pig meat, and 0.04 for cow's milk (Malisch et al., 1996; Pirard and De Pauw, 2005; U.S. EPA, 2005). If the study determined the fat content in food products, these were used for adjustment to fresh weight concentration in lieu of the default values.

For chicken meat, organic chemical content in skin was usually not included by the studies, although skin has a higher fat content and is often consumed with the meat. This would suggest that the skin could have a higher contaminant content than the muscle tissue. Due to lack of skin chemical concentration data and potential loss or destruction of organic chemicals in skin when the meat is cooked, the concentration of chemical in skin was considered similar to the concentration of a chemical in muscle for Tco derivation.

In general, extensive bioaccumulation of persistent, organic chemicals is not as great in either beef or dairy cattle as might be expected, even though beef cattle have no major fat excretion pathway as dairy cattle do with milk production (McLachlan, 1996). This finding is a result of the short life spans and rapid growth dilution that is characteristic of modern animal husbandry. A beef cow develops 100-150 kg of fat in which to deposit the chemical that it absorbs over its 1.5-year life. While a milk cow might excrete its absorbed contaminant in 300 kg of milk fat over the same period, it consumes more feed (and contaminant) in this time. Hence, the chemical concentrations in milk fat were not always much lower compared to beef fat (McLachlan, 1996; RTI, 2005).

Interestingly, the lower-than-expected bioaccumulation of persistent, hydrophobic chemicals in cow's milk does not translate to human milk (McLachlan, 1996). Persistent, organic chemicals tend to bioaccumulate in human milk by an order of magnitude greater than in cow's milk, presuming similar chemical concentrations in the diet on a mg/kg basis. This pronounced difference in bioaccumulation is due to a more limited capability of humans to excrete these chemicals. In addition, the extent of contaminant absorption from food in the human digestive tract may be greater. For example, nursing human infants absorb over 95% of PCBs and most PCDD/Fs while absorption in cows for these same compounds averages closer to 80%.

K.2.1.1 *Diethylhexylphthalate (DEHP)*

At high concentrations (1% DEHP in feed), Tcos for chicken eggs and breast muscle were estimated by OEHHA to be 0.04 and 0.002 d/kg (Ishida et al., 1981; Ishida, 1993). The low transfer values for DEHP relative to other organic chemicals are likely due to rapid metabolism and excretion of DEHP in the chicken.

In dairy cattle, DEHP was observed to be extensively metabolized prior to secretion into the milk (Bluthgen and Ruoff, 1998). OEHHA surmised that much of the metabolism begins in the rumen, where DEHP ester-bond cleavage would occur. Consequently, steady-state is reached in about 7 days and a low milk Tco of 9×10^{-5} d/kg was calculated by OEHHA. Cessation of DEHP administration resulted in nearly undetectable milk levels within 3 days post-exposure. No data could be found regarding residue levels of DEHP in cattle muscle, so a Tco of 4×10^{-4} d/kg was estimated after adjusting for the average fat content difference between cow's milk and cattle muscle. PBPK modeling by MacLachlan (2009) observed a ratio of about 1.5 for residues of highly metabolized lipophilic compounds, such as DEHP, in body fat of non-lactating cows and steers to the same compound in body fat of lactating dairy cows. Thus, the Tco of 4×10^{-4} d/kg was increased by a factor of 1.5 to arrive at a Tco of 6×10^{-4} d/kg for DEHP in meat of beef cattle.

Bioaccumulation data are lacking for DEHP in pigs. Thus, a scaling factor by MacLachlan (2009) was applied for the transfer of lipophilic xenobiotics from lactating cattle to other livestock species. For chemicals such as DEHP that are extensively metabolized in the animal and have a short half-life ($t_{1/2} < 5.8$ d in lactating cows), the ratio of simulated residues in the body fat of pigs to the body fat of lactating dairy cows was essentially equal to 1. Therefore, the dairy cattle muscle Tco determined above (4×10^{-4} d/kg) was only adjusted for the difference in muscle fat content in pig to beef cattle (ratio = 1.2) to arrive at a default Tco of 5×10^{-4} d/kg for pig meat.

K.2.1.2 *Hexachlorobenzene (HCB)*

HCB in the atmosphere is predicted to be predominantly in the vapor phase (Lane et al., 1992). However, due to the extreme persistence of HCB and other chlorinated organic compounds in the environment, deposition and accumulation of non-volatile forms of these organics onto crops, soil and sediment are significant pathways of exposure (Eisenreich et al., 1981; Kelly et al., 1991; Douben et al., 1997; Horstmann and McLachlan, 1998).

In dairy cattle, two studies recorded nearly identical cow's milk HCB Tcos of 0.015-0.016 d/kg with 60-70 days of exposure (Fries and Marrow, 1976; Firestone et al., 1979). The data suggested near steady-state levels in milk were attained with this duration of exposure. A higher Tco of 0.030 d/kg was recorded

in pregnant dairy cattle after about 8 months of exposure (Vreman *et al.*, 1980). Steady-state was reached in milk of the pregnant dairy cattle after about 5 months. The average HCB Tco from these three studies is 0.02 d/kg.

In his review, Kan (1978) provided bioaccumulation data from which to calculate Tcos for HCB. The Tco for egg and chicken muscle were estimated at 16 and 13 d/kg, respectively.

In beef cattle, steady-state levels of HCB were at or near attainment in subcutaneous fat following ten weeks of exposure in the feed (Dingle and Palmer, 1977; RTI, 2005). A muscle Tco estimated from this study was 0.090 d/kg. Exposure to HCB in dairy cattle provided similar Tco values. A muscle Tco of 0.070 d/kg was calculated from HCB concentrations in body fat of lactating dairy cattle following 60 day exposure in the feed (Fries and Marrow, 1976). An eight-month HCB exposure in dairy cattle resulted in a muscle Tco of 0.16 d/kg (Vreman *et al.*, 1980). Because the Vreman study provided a considerably longer exposure overall for cattle, the Tco was based on this study. The PBPK-based scaling factor data by MacLachlan (2009) was applied to estimate the transfer of HCB from lactating cattle to body fat of steers. Using data supplied by Fries and Marrow (1976), a slow elimination half-life of HCB in lactating dairy cattle (average: 50 days) and a small ratio for milk fat concentration over body fat concentration at steady state (0.04) suggests that the PBPK-generated ratio of simulated HCB level in body fat of steers to body fat of lactating dairy cows would be about 1.5. The final default beef Tco is 0.24 d/kg (0.16 d/kg x 1.5)

No data for HCB accumulation in pig muscle tissue could be found. Therefore, a PBPK-based scaling factor was also applied to estimate the transfer of HCB from lactating cattle to pigs (MacLachlan, 2009). The PBPK model results generated a ratio of 0.5 for the simulated HCB level in body fat of pigs to body fat of lactating dairy cows. The final default pig Tco is 0.08 d/kg (0.16 d/kg x 0.5)

K.2.1.3 Hexachlorocyclohexanes (HCH)

HCH Tcos of 7.3 d/kg for egg and 5.1 d/kg for chicken meat were calculated from contaminated feed data provided by Kan (1978) and Szokolay *et al.* (1977). The beta-isomer tended to have roughly 10-fold greater bioaccumulation in poultry egg and muscle than the other major isomers (i.e., alpha and gamma isomers), but is generally found to a lesser extent in the environment. Hence, the Tcos represent a mean of the three major HCH isomers. MacLachlan (2008) developed a model that adequately reproduced the pattern of lindane (gamma-HCH) residue levels in fat and eggs of hens consuming contaminated feed. Utilizing the authors' data, the egg and muscle Tco at steady-state were estimated to be 1.3 and 1.5 d/kg, respectively. These lindane Tcos were similar to those calculated from data by Kan (1978) and Szokolay *et al.* (1977) for eggs, 1.7 and 4.2 d/kg, respectively, and in muscle, 1.8 and 1.2 d/kg, respectively.

As in eggs and meat, the major isomers of HCH (alpha-, beta-, and gamma-HCH), had different patterns of accumulation in cow's milk. The beta isomer has the largest transfer factor, 0.025 d/kg, but generally is in the smallest proportion relative to the other 2 major isomers found in the environment (van den Hoek et al., 1975; Vreman et al., 1976; Vreman et al., 1980). Average Tco values for the alpha- and gamma- (Lindane) isomers were 0.0054 and 0.0014 d/kg, respectively (Williams and Mills, 1964; van den Hoek et al., 1975; Vreman et al., 1980; Surendra Nath et al., 2000). An average Tco for these three HCH isomers is 0.011 d/kg. Surendra Nath et al. (2000) provided data for the industrial grade HCH isomer mixture resulting in a Tco of 0.003 d/kg. The HCH mixture contained 21% gamma-HCH, but further speciation data was not included.

Vreman et al. (1980) fed dairy cows diets containing alpha- and beta-HCH for up to eight months. The calculated muscle Tcos were 0.045 and 0.19 d/kg for alpha- and beta-HCH, respectively. For lindane (gamma-HCH), a Tco of 0.027 d/kg was calculated from a different study following 12-week exposure in non-lactating dairy cattle (Claborn et al., 1960).

We applied a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to beef cattle. Using data supplied by Vreman *et al.* (1980) that showed a cow's milk elimination half-life of 9-19 days for alpha- and beta-HCH, and the data by van den Hoek et al. (1975) that showed similar levels of HCH isomers in milk fat and body fat, the PBPK-generated ratio of simulated HCH levels in body fat of steers to body fat of lactating dairy cows is approximately 2. We multiplied the alpha- and beta-HCH Tcos of 0.045 and 0.19 d/kg, respectively, which were determined in dairy cattle by the scaling factor of 2. The gamma-HCH Tco remained unchanged since non-lactating cows and steers have similar steady state HCH levels in body fat. The average Tco for these three isomers is 0.17 d/kg and is the recommended Tco for beef cattle.

No data for HCH accumulation in pig muscle tissue could be found, so we used a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to pigs. Based on the HCH half-lives and milk fat to body fat ratios in dairy cattle discussed above, the PBPK-generated ratio of simulated HCH levels in body fat of pigs to body fat of lactating dairy cows is very close, or slightly greater, than 1. Thus, Tcos of the three isomers in lactating and non-lactating dairy cows were averaged by us and used as the default for pig meat ($0.045 + 0.19 + 0.027 \text{ d/kg} / 3 = 0.087 \text{ d/kg}$).

K.2.1.4 Polycyclic Aromatic Hydrocarbons (PAH)

Although there are a considerable number of studies investigating PAH exposure in the environment, there are surprisingly few studies that provide reliable data for estimating Tcos in food-producing animals. Exposure of fish, poultry and dairy cattle to a mixture of PAHs results in the presence of mainly low molecular weight PAHs (i.e., three or four cyclic rings) in the fat of meat and milk (Meador

et al., 1995; Grova et al., 2000; Grova et al., 2002; Schaum et al., 2003; Lutz et al., 2006). Many of the high molecular weight PAHs with five or more cyclic rings, such as benzo[a]pyrene (BaP), are known carcinogens or possible carcinogens. Bioaccumulation of PAHs declines with increasing number of aromatic rings and the associated increase in K_{ow} , likely due to both lower gut assimilation efficiency and increased metabolism rate. Another factor appears to be that lower levels of the larger carcinogenic PAHs contaminate pastures and feed compared to the smaller PAHs, often resulting in animal milk and tissue concentrations below the detection limits of analysis equipment (EC, 2002). For example, Muhlemann et al. (2006) found that the larger carcinogenic PAHs in contaminated feed comprised only 8.3% of total PAHs, while the smaller PAHs of four rings or less contributed most of the remaining fraction.

Broiler chickens fed a diet containing low levels of PAHs found in de-inking paper sludge did not exhibit increased PAH levels in abdominal fat for nearly all carcinogenic PAHs examined (Beauchamp et al., 2002). However, the low molecular weight PAHs fluoranthene and pyrene showed increasing levels in abdominal fat with increasing levels of PAHs from paper sludge in the diet of broilers. The carcinogenic potential of these PAHs are undetermined, due to inadequate evidence of carcinogenicity in animals. The calculated broiler muscle Tco for total PAHs was 0.003 d/kg (due mainly to accumulation of pyrene and fluoranthene), and the individual PAH Tcos for pyrene and fluoranthene were 0.1 and 0.04 d/kg, respectively. The total PAH Tco of 0.003 d/kg was chosen as a poultry muscle default value for PAHs, as Tcos for the larger carcinogenic PAHs would likely not surpass this value. No data could be found for PAH accumulation in eggs. Thus, the poultry muscle Tco was also applied to the egg Tco.

The presence of PAHs in milk and milk products suggests that these foods can represent a significant part of human intake of PAHs (Schaum et al., 2003). Among PAHs, the lightest and least lipophilic ones, such as naphthalene, phenanthrene, fluoranthene and pyrene, are detected in the greatest amounts in milk from farms exposed to airborne PAHs (Grova et al., 2000; Grova et al., 2002; Cavret et al., 2005; Lutz et al., 2006). Higher molecular weight PAHs with more than four rings, including possible carcinogens or known carcinogens such as BaP, chrysene and benz[a]anthracene, have been largely undetectable in cow's milk. Of the larger carcinogenic and possibly carcinogenic PAHs, only benz[a]anthracene was detected in tank milk (pooled milk from many cows) sampled near several potential contamination sources (Grova et al., 2002). Levels of this PAH in milk fat ranged from 1.9-2.2 ng/g in milk fat (approximately 0.08-0.09 ng/g in whole milk).

Based on the pasture grass concentrations and corresponding cow's milk concentrations of the three most abundant PAHs (phenanthrene, anthracene, and pyrene) from 10 rural and urban farms investigated by Grova et al. (2000), the range of PAH Tco values in milk were 0.02 to 0.002 d/kg. However, some

assumptions were made to arrive at this estimate, including pasture grass as the only source of ingested PAHs, and intake of pasture grass ranged between 10 to 100% of the cow's diet.

A cow's milk Tco range of 0.002 to 2×10^{-5} d/kg for total PAHs was calculated by OEHHHA from the risk assessment by Muhlemann et al. (2006), based on measurement of total PAHs (roughly 19 PAHs measured) in contaminated feed. Although BaP consisted of only 1.5% of total PAHs, the calculated Tco was within an expected range of 0.013-0.00013 d/kg for BaP. We chose a cow's milk Tco of 0.01 d/kg for total PAHs based primarily on the high-end accumulation of BaP in cow's milk from Muhlemann et al. The recommended Tco is also within the range of 0.02 to 0.002 d/kg estimated for PAHs from data published by Grova et al. (2000).

No data could be found regarding residue levels of PAHs in cattle muscle. The ratio of simulated PAH residues in body fat of steers to body fat of lactating dairy cows for extensively metabolized lipophilic compounds is about 1.4, based on PBPK modeling (MacLachlan, 2009). Assuming equal PAH concentrations in milk fat and body fat of dairy cattle, and application of a scaling factor of 1.4 for dairy cattle to steers, we calculated a default beef Tco for PAHs of 0.067 d/kg ($0.01 \text{ d/kg} \times 0.19/0.04 \times 1.4$).

Accumulation data are also lacking for PAHs in pigs. Using the assumptions from MacLachlan (2009) for transfer of extensively metabolized lipophilic compounds to body fat in livestock, the ratio of PAHs in body fat of pigs to dairy cattle is close to 1. Based on a milk Tco of 0.01 d/kg, adjusting for fat content in pig meat and a scaling factor of 1, we calculate a default pig meat Tco of 0.058 d/kg ($0.01 \text{ d/kg} \times 0.23/0.04 \times 1$).

K.2.1.5 Polychlorinated Biphenyls (PCB)

In dairy cattle, Willett et al. (1990) reviewed early studies that examined the transfer of Aroclor 1254 applied to feed to cow's milk. Tcos of 0.008 to 0.009 d/kg were obtained with doses ranging from 3.5-200 mg/d and exposures ranging from 60-107 days. A cow's milk Tco of 0.01 d/kg for unspciated PCBs from data by Thomas et al. (1999a) was calculated for the sum of 28 PCB congeners found both in feed and the milk.

TEQs have been determined for some PCBs and are generally added to PCDD/F TEQs for calculation of risk assessment values (OEHHHA, 2005). Specific congener Tcos are recommended due to variation in absorption and metabolism of PCBs in dairy cattle, and also due to the degree of chlorination and the position of the chlorine atoms. Some PCBs are transferred effectively unchanged from grass to milk and dairy products (e.g. PCBs 118, 138, 153, 180), with the cow acting as an efficient conduit to humans, while others (e.g. PCBs 52, 101, 149) are largely removed from the environment and the human food

chain if ingested by the dairy cow because they are readily metabolized by the cow (Thomas et al., 1999b). Tcos for individual PCB congeners were estimated from published data and are presented in Table R-1 (Slob et al., 1995; Thomas et al., 1998; Thomas et al., 1999a; Kerst et al., 2004; Huwe and Smith, 2005). Kerst et al. (2004) provided TEQ-adjusted data from which a Tco (WHO-TEQ) of 0.014 d/kg was estimated for unspciated PCBs.

Only one study could be found that allowed development of poultry meat Tcos for a limited number of individual PCB congeners. Pirard and De Pauw (2005) determined bioconcentration factors for coplanar-PCBs (PCBs 77, 81, 126, 169) in chicken breast muscle. Traag et al. (2006) provided bioconcentration data in abdominal chicken fat for all PCBs but exposure lasted only seven days. Because steady-state was not attained, Tcos could not be reliably determined. However, the data do indicate that based on the number of chlorines, the coplanar-PCBs are similarly, or more, bioaccumulative in fat compared to the other PCB congeners with the same number of chlorines. Thus, Tcos for the non-coplanar PCB congeners in Table K-1 were based on the co-planar PCBs with the same number of chlorines.

No reliable data could be found for developing individual congener Tcos for chicken eggs. Thus, the muscle Tcos for individual PCB congeners were also used for eggs, following adjustment for the higher fat content of eggs (11%) compared to muscle (7%).

A general PCB egg Tco of 6.7 d/kg was calculated from a laboratory study in which seven reference congeners (only one of which (#118) is listed in Table K-1) were spiked in the diet of hens (De Vos et al., 2005). Because none of the more bioaccumulative co-planar PCBs were investigated in this study, the coplanar PCB Tco of 10 d/kg was used for unspciated PCBs. Numerous unspciated PCB feed-to-muscle tissue studies have been published in chickens, resulting in a range of Tco values of 2.5 to 7.7 d/kg (Hansen et al., 1983; De Vos et al., 2003; Maervoet et al., 2004; De Vos et al., 2005; Pirard and De Pauw, 2005). A Tco of 7 d/kg for unspciated PCBs was selected as the default value to reflect the median Tco of the individual congeners listed in Table K-1, and because this value is within the range of Tcos for unspciated PCBs.

Toxic equivalent (TEQ)-adjusted Tcos could not be developed for poultry egg or muscle due to lack of steady-state conditions in the studies reviewed. However, a TEQ-adjusted egg Tco of 17 d/kg could be calculated from the bioaccumulation model developed for dioxins and dioxin-like PCBs by Van Eijkeren et al. (2006). Adjusting for the lower fat content in chicken muscle compared to eggs result in a TEQ-adjusted muscle Tco of 11 d/kg.

No reliable data could be found that estimated transfer of PCBs consumed in food to body fat of beef cattle. In dairy cattle, Willett et al. (1990) revised early experiments that examined the transfer of Aroclor 1254 from feed to adipose

tissue. Fresh weight beef Tcos of 0.013 to 0.027 d/kg were obtained for doses ranging from 10-200 mg/d with 60 day exposures. In another study, a beef Tco of 0.024 d/kg was calculated for dairy cattle following 14-week consumption of PCBs that naturally contaminated pastures (Thomas et al., 1999a).

On a fat weight basis, Thomas et al. (1999b) observed that not only are the PCB concentrations in body fat and milk fat similar, but that the congener patterns were similar as well. Thus, even though comprehensive congener-specific data are lacking for PCBs in muscle, congener-specific beef Tcos can be estimated from the cow's milk Tco data by adjusting for the greater fat content in muscle tissue (19%) compared to the milk fat content (4%).

We applied a PBPK-generated scaling factor developed by MacLachlan (2009) to estimate the transfer of PCBs from body fat of lactating cattle to body fat of beef cattle. Using data by Huwe and Smith (2005) that found a cow's milk half-life of 39-196 days for some co-planar PCBs, and the data by Thomas et al. (1999b) that showed similar levels of PCBs in milk fat and body fat, the ratio of simulated co-planar PCB levels in body fat of steers to body fat of lactating dairy cows is approximately 10. We multiplied the scaling factor of 10 to the PCB milk Tcos in Table K-1 following adjustment for differences in fat content between milk and beef to generate Tcos for beef. A similar calculation was also used to estimate the TEQ-adjusted beef Tco for PCBs.

In swine, Arochlor 1254 was added to feed for 6 months resulting in an unspciated PCB Tco of 0.52 d/kg (Hansen et al., 1983). Speciated Tcos for 16 PCBs could be determined from the data, although only one PCB (#118) is currently listed in Table K-1. Thus, Tcos for individual PCBs in Table K-1 were based on the highest calculated PCB Tco with the same number of chlorines from the Hansen et al. study. No information could be found for a TEQ-adjusted Tco for PCBs in swine, so the Tco was based on a Tco of 0.07 d/kg for meat of dairy cattle. The TEQ-adjusted value was then adjusted using a ratio of 3 for PCBs in body fat of pigs to body fat of lactating dairy cows from MacLachlan (2009), and then accounting for the higher average fat content in pig meat (23%) compared to cattle (19%). The default TEQ-adjusted pig Tco is 0.25 d/kg.

K.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Furans (PCDD/F)

Numerous studies have investigated the feed-to-cow's milk transfer of PCDD/Fs. Several of these studies were conducted in the field near municipal solid waste incinerators, or estimated the mass balance of PCDD/F intake resulting from exposure to background or elevated levels of PCDD/Fs in pasture and soil (McLachlan et al., 1990; Slob et al., 1995; Schuler et al., 1997b; McLachlan and Richter, 1998; Lorber et al., 2000). These types of studies likely represent the best data for developing individual congener and overall unspciated transfer factors of PCDD/Fs from "Hot Spots" facilities. Averaged congener Tco values were estimated from these data and are presented in Table K-1.

The milk Tco decreases by an order of magnitude or more for some of the higher chlorinated PCDD/Fs. This trend agrees with models showing that the percent transfer of chemical from feed to milk decreases for compounds with log Kow larger than about 6.5 (McLachlan, 1996). This reduced absorption is attributed to the presence of an aqueous resistance that limits diffusion of very hydrophobic compounds through the intestinal wall. Thus, a Tco for total PCDD/Fs (unspeciated PCDD/Fs) has not been pursued by researchers in their exposure studies. Nevertheless, a Tco for unspeciated dioxin-like PCDD/Fs of 0.001 d/kg can be calculated from the data by McLachlan et al. (1990). In addition, an averaged TEQ-adjusted (I-TEQ) Tco value of 0.005 d/kg could also be calculated from the data of Slob et al. (1995) and McLachlan et al. (1990).

Several studies provided data from which Tcos could be estimated for individual PCDD/F congeners found in eggs and chicken meat. For eggs, transfer factor data were derived from three studies in which feed was mixed with soil environmentally contaminated with PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Schuler et al., 1997a), and one study of feed contaminated with fly ash (Pirard and De Pauw, 2006). Individual congener Tcos among the studies were similar, often within a factor of five between values. An average Tco was calculated for each congener from the four studies and is shown in Table K-1.

Many of the same studies in chickens also estimated accumulation values for the sum of all PCDD/F congeners, or unspeciated PCDD/Fs, in eggs and meat. In egg, four studies in free-range and laboratory chickens exposed to contaminated soil provided an average Tco of 5.5 d/kg (range: 1.9 to 13.1 d/kg) for unspeciated PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Malisch et al., 1996; Schuler et al., 1997a). In chicken muscle, three contaminated feed or soil studies provided accumulation data from which an average Tco of 4.6 d/kg (range: 1.0 to 7.6 d/kg) was calculated (Stephens et al., 1995; Iben et al., 2003; Pirard and De Pauw, 2005).

However, unspeciated PCDD/F accumulation was most often expressed in toxic equivalents (TEQs). Three controlled laboratory studies in which 10% of the diet was PCDD/F-contaminated soil, the calculated TEQ-adjusted Tcos for eggs ranged from 2.4 to 4.1 d/kg with an average of 3.6 d/kg (Petreas et al., 1991; Stephens et al., 1995; Hoogenboom et al., 2006). For the controlled laboratory feed-to-egg studies in which PCDD/Fs in fly ash or oil were added to feed (i.e., no contaminated soil was added to the diet), egg Tcos ranged from 8.5 to 17 d/kg with a mean of 12 d/kg (Pirard and De Pauw, 2005; 2006; Van Eijkeren et al., 2006).

For field studies, calculated egg Tcos of free-foraging chickens in various regions with PCDD/F-contaminated soil showed greater variation and was higher (Schuler et al., 1997a; Harnly et al., 2000; Hoogenboom et al., 2006). The Tcos ranged from 12 to 37 d/kg with an average of 23 d/kg. An assumption was made

that the PDCC/F source for the free-foraging hens was contaminated soil, and that the soil ingestion rate was 10 g soil/day. There is general support among researchers for this soil ingestion rate by free-foraging chickens (De Vries et al., 2006). The larger egg Tco in field studies compared to controlled laboratory studies may be a result of free-foraging chickens consuming soil organisms and herbs and grass which may also be contaminated. However, greater bioavailability of soil PCDD/Fs in the field, or a higher soil ingestion rate than predicted may also play a role in a larger egg Tco under field conditions.

Overall, the range of mean values for these three types of studies is not large (within a factor of 10), considering the different sources of PCDD/Fs that the poultry were exposed to. A grand mean from the three types of exposure studies (contaminated soil field study, controlled contaminated soil study and contaminated feed study) is 13 d/kg ($3.6 + 23 + 12 \text{ d/kg} / 3$), which we recommend as the default egg Tco for PCDD/Fs.

For edible muscle tissue (usually thigh or breast tissue), TEQ-adjusted Tcos could be calculated from several studies that investigated PCDD/F concentrations in chickens given contaminated feed. In a controlled laboratory study in which 10% of the diet was PCDD/F-contaminated soil, a Tco of 7.4 d/kg was calculated (Stephens et al., 1995). In three contaminated feed studies where PCDD/Fs in oil or fly ash were added to diet, similar Tcos of 8.6, 9.0 and 4.1 d/kg were calculated (Iben et al., 2003; Pirard and De Pauw, 2005; 2006). A mean of 7.3 d/kg is calculated from these data and represents the TEQ-adjusted poultry meat Tco for PCDD/Fs.

Congener-specific data for development of beef Tcos was not as comprehensive as that for development of cow's milk Tcos. Two long-term pentachlorophenol (PCP) feeding studies in dairy cattle determined body fat concentrations for several PCDD/F congeners (1, 2, 3, 6, 7, 8- and 1, 2, 3, 7, 8, 9-HxCDD, 1, 2, 3, 4, 6, 7, 8-HpCDD, OCDD, 1, 2, 3, 4, 6, 7, 8-HpCDF, and OCDF) that were contaminants in the PCP formulation (Firestone et al., 1979; Parker et al., 1980). Beef Tcos based on dairy cattle for the other congeners and unspciated PCDD/Fs were estimated with the assumption that the fat concentration is similar in milk and beef, and were adjusted upward to account for the greater fat content in muscle tissue (19%) compared to the fat content in milk (4%). As noted above, the concentration of PCBs in milk fat and body fat have been shown to be similar in exposure studies (Thomas et al., 1999b). We then applied scaling factors by MacLachlan (2009) to estimate the transfer of PCDD/Fs from body fat of lactating cattle to body fat of beef cattle. Data by Huwe and Smith (2005) found half-lives were mostly 30-50 days for the PCDD/Fs; the major exceptions were OCDF ($t_{1/2} = 14$ days) and OCDD ($t_{1/2} = 72.6$ days). A ratio of 7 is estimated for the simulated PCDD/F levels in body fat of steers to body fat of lactating dairy cows for most PCDD/Fs. A ratio of 4 was estimated for OCDF and a ratio of 10 was estimated for OCDD.

Pig Tcos for individual and unspeciated PCDD/Fs in Table K-1 were estimated from a comprehensive study in which PCDD/Fs were added to the diet in feed of pigs during the 12-week fattening period (Spitaler et al., 2005). This exposure period represents the last 12-weeks prior to slaughter in the typical 6-month life of a pig. Notably, the researchers did not observe a reduction of residues due to roasting of the meat.

K.2.2 Tcos for Inorganic and Organic Metals

The studies used to derive inorganic and organic metal Tcos listed in Table K-2 usually presented data as fresh weight concentrations in muscle, milk and eggs. Occasionally, dry weight concentrations were reported. Unless the study noted the water content of the food source, default factors of 0.87 for cow's milk, 0.35 for chicken egg, 0.25 for chicken meat, and 0.30 for beef and pork were used for adjusting to fresh weight concentration (USDA, 1975).

Biotransfer studies for pig muscle could not be found for most of the metals. As noted in the beginning of this appendix, biotransfer data in cattle was more abundant. Where specific metal biotransfer data were missing in pigs but present in cattle, the pig meat Tco was estimated using a simple metabolic weight adjustment from cattle to pig as shown in Eq. K-1.

In general, low concentrations of inorganic metals are transferred from contaminated feed to muscle tissue, cow's milk and eggs and are not as great a concern relative to other potential sources of heavy metals in multipathway exposures. However, many of the inorganic metals such as cadmium, lead and mercury tend to accumulate over time in organs, particularly kidney and liver. Thus, frequent consumption of organs from exposed food animals may present a much greater toxic hazard to humans than consumption of the meat. Cadmium is of particular concern due to its relatively high toxicity and high potential for accumulation in the kidney and liver. Kidney and liver-specific Tcos for cadmium and a few other metals are presented in the text below for some of these food-producing animals only for comparison purposes. Tcos for accumulation in bone for some of the metals (i.e., lead) are also noted or calculated for some of the food products.

K.2.2.1 Arsenic

Only one study could be located that recorded a measurable increase of arsenic in cow's milk following dairy cattle consumption of contaminated feed. We calculated a Tco of 5×10^{-5} d/kg from data in dairy cattle exposed to As(III) as arsenic trioxide for 15-28 months (Vreman et al., 1986).

In poultry, organic arsenic compounds are an approved dietary supplement that can result in increased levels of total arsenic in meat and eggs (Lasky et al., 2004). Both organic and inorganic forms of arsenic are found in poultry, with

inorganic forms more toxic than organic forms. Analysis of poultry and meat samples indicate that about 65% of total arsenic is in the inorganic form.

We calculated a Tco of 0.07 d/kg for total arsenic in eggs from hens fed a diet containing arsenic trioxide (Holcman and Stibilj, 1997). In muscle, total arsenic Tcos of 0.06 and 0.02 d/kg were determined in chickens from two studies following addition of arsenic trioxide to feed (Overby and Frost, 1962; Vadrnjak et al., 1997). The proportion of arsenic in the inorganic form was not determined. In drinking water, soluble As(V) was added to the water resulting in a total arsenic Tco of 0.2 d/kg in muscle of broiler chickens (Pizarro et al., 2004). However, only 10% of arsenic in muscle was in the inorganic form. Over 50% was present as dimethylarsinic acid, which is considered a methylation detoxification pathway for arsenic. Thus, the inorganic arsenic Tco was 0.02 d/kg. We calculated an average muscle Tco of 0.03 d/kg from the three studies for transfer of arsenic from diet to chicken meat.

In beef cattle, Vreman et al. (1988) administered arsenic trioxide in the feed for 143 days to 16 bulls at about 12.5 mg/d resulting in a muscle Tco of 2.4×10^{-3} d/kg. The same Tco was calculated from data by Ham et al. (1949) that dosed adult steers daily with 270 mg arsenic trioxide for 201 days. In another study in steers, Bruce et al. (2003) estimated the daily intake of arsenic from grazing pasture grass, ingesting dust adhering to pasture, and direct ingestion of soil in an area contaminated with arsenic-laced mine tailings. Based on the daily intake and muscle concentration of arsenic at sacrifice after 237 days of exposure, a Tco of 2.8×10^{-4} d/kg was derived. We calculated an average muscle Tco of 1.7×10^{-3} d/kg from these three studies, which we recommend as the default value for beef cattle. Long-term arsenic feeding studies have also been conducted in lactating dairy cows. A slightly lower muscle Tco of 7.1×10^{-4} d/kg was calculated from these studies (Peoples, 1964; Vreman et al., 1986).

Arsenic exposure in beef and dairy cattle has not shown tissue-specific sequestering in liver or kidney, unlike some of the inorganic metals (e.g., cadmium, lead, and mercury). Similar Tcos were estimated for muscle, liver and kidney (Ham et al., 1949; Peoples, 1964; Vreman et al., 1986).

K.2.2.2 *Beryllium*

No inorganic beryllium accumulation studies could be found in the literature for poultry. Thus, we calculated poultry egg and meat Tcos for beryllium based on the average Tco value of the other "Hot Spots" divalent, cationic metals in Table K-2 (i.e., cadmium, lead, inorganic mercury, and nickel) providing a beryllium Tco for egg and muscle of 0.09 and 0.2 d/kg, respectively.

No multiple day inorganic beryllium exposure studies have been conducted in cattle or swine. In a single bolus study, Ng (1982) estimated a cow's milk Tco of 9.1×10^{-7} d/kg based on recovery of radiolabeled beryllium chloride given to dairy

cattle. For beef, we determined a beryllium Tco of 3×10^{-4} d/kg based on the average Tco value of the divalent, cationic metals cadmium, lead, and inorganic mercury. Beef Tcos for these three metals were determined directly from published studies. A default pork Tco was determined by us by the same method as that used for beef, resulting in a pig meat Tco of 1×10^{-3} d/kg.

K.2.2.3 *Cadmium*

Very low accumulation of cadmium occurs in cow's milk, and concentrations of cadmium in cow's milk are often below the detection limit. In his review, Stevens (1991) estimated an average Tco of 1.3×10^{-6} d/kg in cow's milk from two long-term cadmium exposure studies by Vreman et al. (1986). More recently, we estimated a milk Tco of 1.3×10^{-5} d/kg from exposure data in a single cow exposed to cadmium for 77 days (Mehennaoui et al., 1999). The average Tco from the three exposure studies is 5×10^{-6} d/kg, which we recommend as a default Tco.

Numerous cadmium accumulation studies have been conducted in poultry. Similar to cow's milk, very low accumulation of cadmium occurred in hen's eggs with exposure in feed; the levels of cadmium in eggs are sometimes below the detection limit. We calculated an average egg Tco of 0.01 d/kg from the best available data (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985). In muscle, we determined cadmium Tcos in exposed chickens ranging from 0.2 to 1 d/kg (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985; Pribilincova et al., 1995; Bokori et al., 1996). The average value from these studies was 0.5 d/kg, which we recommend as the Tco.

Similar cadmium Tcos in muscle of dairy and beef cattle have been observed in long-term feeding studies lasting 3.5 to 28 months. We calculated an average Tco of 2.0×10^{-4} d/kg with a range of $1.2 - 3.2 \times 10^{-4}$ d/kg (Johnson et al., 1981; Vreman et al., 1986; 1988). A muscle Tco of 6.5×10^{-5} d/kg was obtained from a feeding study by Lamphere et al. (1984) describing cadmium body burden in calves exposed for 60 days. However, the short exposure duration only during growth of the animal may result in an underestimation of the Tco compared to exposure to adulthood.

Cadmium accumulates to a much greater extent in some organs compared to muscle tissue. In poultry, exposure studies suggest that cadmium accumulation in the kidney and liver increases with increasing exposure duration and may not attain a steady-state concentration. Eighty-week exposure to cadmium in chickens resulted in a Tco of 800 d/kg in the kidney and 70 d/kg in the liver (Hinesly et al., 1985). In dairy and beef cattle, cadmium Tcos for liver and kidney did not vary greatly even though exposure durations varied. Average calculated Tcos were about 0.03 d/kg (range: 0.01 to 0.048 d/kg) for liver, and 0.1 d/kg (range: 0.09 to 0.19 d/kg) for kidney (Sharma et al., 1979; Sharma et al., 1982; Vreman et al., 1986; 1988).

Only one study could be found that measured cadmium muscle levels in pigs following exposure to cadmium in feed. Cousins et al. (1973) only found measurable cadmium levels in skeletal muscle at the highest of four doses tested (1350 ppm) following six-week exposure, but this level caused severe toxicity. More accurate estimates of muscle uptake were found in heart tissue, which exhibited increased tissue concentration with increasing dose and may represent the upper end of the cadmium concentration found in skeletal muscle. The average Tco we calculated in heart muscle was 0.0051 d/kg. In the liver and kidneys of pigs, cadmium Tcos as high as 0.48 and 2.53 d/kg, respectively, were calculated from a study by Sharma et al. (1979).

K.2.2.4 Chromium (Hexavalent)

Only a portion of ingested hexavalent chromium (Cr(VI)), perhaps 1-2%, is expected to be systemically absorbed in the hexavalent form due to rapid reduction to the less soluble and less toxic trivalent chromium in the acidic environment of the stomach (Costa, 1997; NTP, 2008). Trivalent chromium (Cr(III)) is an essential micronutrient, but no cancer potency or noncancer reference exposure level is currently available for this form of chromium. Cr(VI) that is absorbed can then be actively transported into all cells and tissues of the body in place of anions, such as phosphates. Once inside the cell, the Cr(VI) is reduced to various unstable reactive intermediates and, finally, stable Cr(III) is ultimately formed inside the cell.

Current analytical procedures cannot differentiate between the oxidation states of chromium in biological tissues (NTP, 2008). However, it has been advocated that any Cr(VI) transported into meat and eggs would be converted to the more stable Cr(III) form and would presumably not pose a risk for human consumption (Chundawat and Sood, 2005). Based on these findings no Cr(VI) Tco is currently recommended by OEHHA for meat and eggs.

However, a similar situation may not be the case for cow's milk. Lameiras et al. (1998) found Cr(VI) in cow's milk, which was 2-4 times lower compared to total chromium. In whole milk, the average total chromium concentration was 2.70 ug/L (range: 1.42-5.70 ug/L) and the average Cr(VI) concentration was 0.68 ug/L (range: 0.20-1.20 ug/L). No multiple day Cr(VI) exposure studies in dairy cattle could be found in the literature. Following a single oral dose of radiolabeled sodium chromate (Na_2CrO_4), Van Bruwaene et al. (1984) calculated a steady-state cow's milk Tco of 1.0×10^{-5} d/kg for total chromium. Stevens (1991) estimated a similar Tco of 1.4×10^{-5} d/kg from the same data based on a half-life of 26 days for total chromium in cow's milk. These studies did not attempt to estimate the proportion of total chromium that was secreted as Cr(VI) into milk.

Multiplying the Stevens total chromium Tco by the fraction of total chromium that is Cr(VI) in normal milk (1.4×10^{-5} d/kg \times 0.68/2.70 ug/L) provided a modified Tco

of 3.5×10^{-6} d/kg. Until valence-speciated cow's milk data is available from Cr(VI) exposure studies, we chose a midpoint Tco value between the Stevens Tco and this modified Tco adjusted for Cr(VI) content in normal milk (8.75×10^{-6} d/kg) as a health-protective cow's milk default value for Cr(VI).

K.2.2.5 Fluoride

In a series of long-term exposure studies on fluorides' effect on milk production, the fluoride concentration in the milk of dairy cows given fluoride in feed resulted in an estimated cow's milk Tco of 0.0003 d/kg (Stoddard et al., 1963; Harris et al., 1964).

Fluoride in the diet of hens resulted in very low accumulation of fluoride in muscle, and yolk and albumin of eggs (Hahn and Guenter, 1986). We calculated a Tco in whole eggs of 0.008 d/kg from the exposure data. Considerably greater accumulation occurs in egg shell. Muscle accumulation in the fluoride-exposed hens resulted in a Tco of 0.03 d/kg.

Specific data concerning accumulation of fluoride in the skeletal muscle tissue of exposed cattle could not be found. However, in cases of high fluoride intake, fluoride levels in the soft tissue (i.e., brain, liver, kidney, pancreas, intestines, etc.) are reported to increase only two or three times the normal value in meat producing animals. Fluoride does not accumulate in the edible portions of the animal (Suttie et al., 1958; Shupe et al., 1964). However, considerably greater accumulation of fluoride occurred in bone. In heart tissue, we calculated a fluoride Tco of 8.4×10^{-4} d/kg for Holstein cows fed fluoride-contaminated rations for 5.5 years, which we recommend as the default muscle Tco for range cattle (Suttie et al., 1958). It is assumed that similar pharmacokinetic properties, and similar Tcos, occur for fluoride in both skeletal and heart muscle tissue.

K.2.2.6 Lead

Only three contaminated feed studies observed measurable levels of lead in milk from both control and exposed dairy cows. Based on data from a 15-28 month lead exposure study of dairy cows kept indoors, a cow's milk Tco of 2.6×10^{-5} d/kg was calculated (Vreman et al., 1986). A three-month outdoor lead exposure study by the same researchers produced a Tco of 5.4×10^{-5} d/kg. Stating that the half-life of lead in dairy cows is about 45 days, Stevens (1991) adjusted the Tco of the three-month outdoor study to 7.1×10^{-5} d/kg. However, Willett et al. (1994) observed that steady-state was attained in cow's milk after only 14 days of a 49-day lead exposure study, generating a Tco of 7.9×10^{-5} d/kg. Using the steady-state-corrected Tco by Stevens (1991) for the outdoor Vreman study, we recommend an average Tco of 5.9×10^{-5} d/kg from these three studies.

An average Tco of 0.4 d/kg in muscle was calculated by OEHHHA for lead in broiler chicks fed contaminated feed for 20 days (Stoddard et al., 1963; Harris et

al., 1964; Latta and Donaldson, 1986a; 1986b). For comparison, a roughly 10-fold higher Tco was calculated for lead in kidney. However, lead tends to accumulate most in bone, generating a Tco of 70 d/kg. Lead in bone is not expected to be a problem, unless contaminated bone is ground into bone meal and fed to animals. Accumulation of lead in eggs was very low, generating a Tco of 0.04 d/kg (Meluzzi et al., 1996).

Vreman et al. (1988) administered lead acetate in feed to young bulls for 143 days during the fattening period. The resulting muscle Tco was 2.7×10^{-4} d/kg. A slightly lower muscle Tco of 6.7×10^{-5} d/kg in lactating dairy cows fed lead mixed with their feed (Vreman et al., 1986).

Roughly 10- to 100-fold greater accumulation of lead occurs in the kidney and liver of cattle compared to their muscle tissue. We calculated Tcos of 4.8×10^{-3} and 1.4×10^{-2} d/kg for liver and kidney, respectively, in the bulls from the Vreman et al. (1988) study. In addition to liver and kidney, lead was also found to accumulate in bone. In a three-month feeding study in dairy cattle, a bone Tco of 0.02 d/kg was calculated from the data by Sharma et al. (1982). In one of the few biotransfer studies conducted in pigs, a liver Tco of 1.4×10^{-2} d/kg was recorded in pigs fed diets containing either 5 or 25 ppm lead acetate for 90 days (Sharma and Street, 1980).

K.2.2.7 *Mercury (Inorganic and Methyl Mercury)*

Addition of only inorganic mercury (Hg(II)) to the feed of hens for 140 days resulted in a muscle tissue Tco of 0.02 d/kg (Kiwimae et al., 1969). However, some Hg(II) was converted to methyl mercury (MeHg) in the chickens, resulting in a muscle Tco of 0.09 d/kg for MeHg. When only MeHg is added to the diet in prolonged feeding studies, an average Tco of 10 d/kg was calculated with virtually all the mercury in the muscle as MeHg (Kiwimae et al., 1969; Soares et al., 1973; Hilmy et al., 1978). Some Hg(II) added to feed is also endogenously methylated in the hens and transported to the eggs. Addition of Hg(II) to the feed of hens for 140 days resulted in a calculated egg Tco of 0.3 d/kg for Hg(II), and 0.5 d/kg for MeHg (Kiwimae et al., 1969). An average egg Tco of 11 d/kg was calculated when only MeHg was added to feed (Scott et al., 1975; Hilmy et al., 1978).

Vreman et al. (1986) observed a small, but statistically insignificant increase in mercury in cow's milk with exposure of dairy cattle to inorganic mercury in feed for 15-28 months. The Tco range was 7 to 40×10^{-5} d/kg with an average of 2×10^{-4} d/kg. Stevens (1991) calculated Tcos of 9.2×10^{-6} and 1.3×10^{-5} d/kg from oral single bolus studies of radiolabeled inorganic mercury by Mullen et al. (1975) and Potter et al. (1972). The steady-state Tcos were calculated by use of study-specific half-lives of 1.2 (Potter et al., 1972) or 5.5 days (Mullen et al., 1975) for mercury. We calculated an average Tco of 7×10^{-5} d/kg from the three studies, which we recommend for transfer of inorganic mercury to cow's milk.

Similar to cow's milk, only a small, but statistically insignificant increase in inorganic mercury could be measured in muscle tissue following long-term exposure of dairy and beef cattle to soluble mercury (Vreman et al., 1986; 1988). Calculated maximum muscle Tco values from these two studies were $6.7\text{--}18 \times 10^{-4}$ d/kg, but we lack confidence in this value due to the detection limit of these studies. To calculate the biotransfer of ingested mercury to muscle, Stevens (1992) relied on three oral bolus dose studies that determined the half-life of inorganic mercury in blood of dairy cattle (Potter et al., 1972; Ansari et al., 1973; Mullen et al., 1975). Operating on a reasonable assumption that muscle is a well-perfused tissue and shares the same kinetic compartment as blood, Stevens calculated an average muscle Tco of 3.5×10^{-4} d/kg (range: $1.8\text{--}4.4 \times 10^{-4}$ d/kg). This value is comparable with the Tcos estimated from the Vreman studies and we recommend as the point estimate Tco for inorganic mercury in beef.

Although it is not anticipated that human exposure to methyl mercury via cow's milk and beef would be a significant pathway (compared to fish), biotransfer information is included here for completeness. There are few published data that investigated ruminant methylmercury uptake and accumulation. However, background exposure and accumulation of inorganic and methylmercury in meat products is reported to be very low (U.S. EPA, 1997). In their risk assessment guidelines, U.S. EPA (2005) suggests that only 13% of total mercury in ruminants is present as methylmercury, an indication that ruminants have little exposure to methylmercury.

In vitro, cow rumen microflora does not methylate added inorganic mercury (as HgCl_2) to methylmercury (Kozak and Forsberg, 1979). On the other hand, rumen microflora was found to demethylate up to 40% of added methylmercury to metallic mercury (Hg^0), which would then be presumably excreted with little or no absorption. This finding suggests that ruminants can detoxify some of the ingested methylmercury.

Stevens (1991) estimated that the Tco for methylmercury in cow's milk is roughly one order of magnitude greater than that for inorganic mercury (i.e., 7×10^{-4} d/kg). His finding was based on a study by Neathery et al. (1974), in which two dairy cows were given a bolus dose of radiolabeled methylmercuric chloride and followed for the appearance of label in milk for 14 days. A milk excretion half-life of 6 days was calculated from the data. It was suspected that the lipophilic nature of methyl mercury resulted in its accumulation in milk fat. Of the labeled methylmercury that was absorbed, 72% of the total body burden was found in muscle tissue 15 days after the single bolus dose. However, there are insufficient data to estimate the biotransfer of ingested methylmercury in cattle and pigs with chronic exposure.

K.2.2.8 *Nickel*

Only two studies were found in the literature that attempted to estimate the nickel concentration in cow's milk following 1.5 to 2 month exposure of the dairy cattle to inorganic nickel-contaminated feed (Archibald, 1949; O'Dell et al., 1970). Neither study used analysis methods that were sensitive enough to record measurable increases of nickel in the cow's milk. Stevens (1991) used the maximum value approach by dividing the detection limit (0.1 ppm) of the studies by two, arriving at an average cow's milk Tco of 2.7×10^{-5} d/kg. Until more sensitive studies are conducted, we recommend this Tco as the default value for inorganic nickel.

Limited data for nickel indicate low accumulation of this metal occurs in eggs and tissues of chickens (Ling and Leach, 1979; Meluzzi et al., 1996). We calculated Tcos of 0.02 d/kg for both eggs and muscle tissue of hens fed inorganic nickel mixed in their diet. As with other inorganic metals, greatest nickel accumulation occurred in the kidney (Tco = 0.68 d/kg), resulting in a Tco over 30-fold higher than that found in muscle or eggs.

No adequate studies investigating biotransfer of ingested inorganic nickel to beef or pork could be located. As with the approach used for beryllium, we determined a beef Tco based on an average of the three divalent cationic metal Tcos (i.e., cadmium, lead and inorganic mercury) that had sufficient biotransfer data available in the literature. The resulting beef Tco was 3×10^{-4} d/kg. We then developed a pig meat Tco of 0.001 d/kg based on the cow-to-pig metabolic weight ratio adjustment (Eq. K-1). OEHHA recognizes that these Tcos developed for beef and pork are more uncertain than would be desirable. However, the data available in other food-producing animals and similar Tcos developed for other cationic metal contaminants indicates the nickel muscle Tco is likely not underestimated in cattle and pigs.

K.2.2.9 *Selenium*

The selenium concentration in milk tends to increase as intake of selenium increases from about 2 to 6 mg/day (Fisher et al., 1980; Maus et al., 1980; Beale et al., 1990). Secretion of selenium into milk then appears to reach a temporary limit when selenium intake is about 6 to 12 mg/day. The mammary gland is either limited in the limited amount of selenium it can secrete into milk, or, more likely, the net absorption of selenium from the gut is controlled in the face of increased selenium intake. Only when selenium intake increases above 50-100 mg/day does the ability of the protection mechanism become exceeded, resulting in selenium toxicity and increased selenium concentration in milk. We calculated a Tco of 0.009 d/kg based on the average value for studies that supplemented feed with 6 mg/d selenium or less.

Optimum levels of selenium in the diet of poultry are about 0.1 to 0.2 ppm (Arnold et al., 1973; Moksnes and Norheim, 1982). Concentrations of selenium above 3 ppm may result in toxicity. At concentrations of 1 to 9 ppm selenite in the feed, we calculated an average egg Tco of 3 d/kg (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982; Davis and Fear, 1996). In broiler chicks, an average Tco of 0.9 d/kg for muscle was calculated (Moksnes and Norheim, 1982; Echevarria et al., 1988a; 1988b). Laying hens had a lower Tco of 0.4 d/kg for muscle tissue, possibly due to eggs acting as an elimination pathway for selenium (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982). Thus, the muscle Tco for selenium is based on the findings in meat (broiler) chickens.

In beef cattle, groups of calves were fed sodium selenite in a milk replacer at concentrations of 0.2 to 5 ppm for six weeks (Jenkins and Hidiroglou, 1986). We calculated an average muscle Tco of 6.6×10^{-2} d/kg from the exposure data. In another study, inorganic selenium was intraruminally administered in beef cows through two soluble-glass boluses to slowly release Se over approximately 11 months (Hidiroglou et al., 1987). We calculated a Tco of 7.1×10^{-3} d/kg in the muscle tissue. The average muscle Tco from the two studies is 0.037 d/kg, which we recommend as the default selenium transfer factor. Jenkins and Hidiroglou (1986) also observed greater accumulation of selenium in the liver and kidney cattle compared to muscle, resulting in calculated Tcos of 2.7 and 0.25 d/kg, respectively.

In pigs, selenium muscle concentrations have been measured following unsupplemented intake or supplementation of selenium in diets. No studies could be located that estimated tissue levels of selenium following prolonged intake of toxic or near-toxic levels of selenium. Using a study by Ku et al. (1972), we calculated an average muscle Tco of 0.61 d/kg in groups of adult pigs that had been fed diets containing selenium at levels ranging from 0.027 to 0.493 ppm. A positive correlation between selenium level in the diet and muscle concentration was observed. Using another study, which exposed pigs to diets containing 0.78-0.88 ppm selenium during the growth phase, we calculated a muscle Tco of 0.35 d/kg in pigs at market weight (Jenkins and Winter, 1973).

Similar to the phenomena observed in dairy cattle, supplementation of pig diets with selenium (0.1 to 1.0 ppm) did not always result in an increase in tissue selenium levels. Tcos based on these studies are as much as 10-fold lower compared to Tcos calculated from baseline levels of selenium found in feed (Groce et al., 1971). However, it is not known if this protective mechanism also operates at higher selenium levels in feed that may produce toxic effects in pigs. Thus, we recommend a default pig Tco based on the average Tco (0.48 d/kg) determined using Ku et al. (1972) and Jenkins and Winter (1973), which covered a range of baseline selenium intakes in feed from 0.027 to 0.88 ppm.

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Appendix L

Activity Data Analysis Report

L.1 Introduction

The Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) staff are evaluating ways to update and improve exposure assessment methodologies and the data used for conducting Health Risk Assessments (HRA) as prescribed under the Air Toxics "Hot Spots" Information and Assessment Act (Assembly Bill 2588; Health and Safety Code Section 44300 et seq.). The goals of the Air Toxics "Hot Spots" Act are to collect emission data, to identify facilities having localized impacts, to ascertain health risks, to notify nearby residents of significant risks, and to reduce those significant risks to acceptable levels. This report focuses on the two exposure variables (i.e. exposure duration and exposure frequency) used in estimating a person's lifetime average daily dose by considering the time a person lives in their primary residence and the time they spend daily at home. Past OEHHA HRA methodologies and various programs utilize residency periods of 9, 30, and 70 years.

Staff looked into various data sources to determine the residency duration at the household level and the daily activity pattern at the individual level. The data sources the staff examined include the National Human Activity Pattern Survey (NHAPS), the National Household Travel Surveys (NHTS), the National Longitudinal Surveys, the American Time Use Survey Data Extract Builder, the Integrated Public Use Microdata Series (IPUMS-USA) census data, the Southern California Association of Governments (SCAG) 2000 regional travel survey data, and the California Department of Transportation (Caltrans) 2000-2001 California Statewide Household Travel Survey (CHTS) data. The staff determined that IPUMS-USA, SCAG 2000 regional travel survey, and Caltrans 2000-2001 CHTS represent the most current and California-specific residence and activity data and therefore were used as the basis for the conclusions stated in this report.

Results show that, from 2006 to 2009, over 91% of California householders had lived at their current home address for less than 30 years, and over 63% of householders had lived at their current residence for 9 years or less. No data was available for householders who lived in their homes over a 70 year period.

Exposure frequency adjustments (e.g. number of days per year) are also used in current HRA methodology. The 2000-2001 CHTS data shows that, on average, Californians spend approximately 73% of their time at home per day. When looking at the data by age group, the time increases to 85% for children under 2 years old. Individuals that are 2 years or older, but less than 16 years old, spend 72% of their time at home; whereas Californians that are 16 years or older spend 73% of their time at home.

L.2 Data Sources Analyzed

L.2.1 IPUMS-USA data

IPUMS-USA consists of more than fifty samples of the American population drawn from fifteen federal censuses and from the American Community Surveys (ACS). ACS is a nationwide survey that collects and produces population and housing information every year from about three million selected housing unit addresses across every county in the nation (ACS). IPUMS-USA samples, which draw on every surviving census from 1850-2000 and the 2000-2009 ACS samples, collectively constitute the quantitative information on long-term changes in the American population. These records for the period since 1940 only identify geographic areas with equal or larger than 100,000 residents (250,000 in 1960 and 1970) (IPUMS-USA).

IPUMS-USA census data contains residency duration, travel to work data, residence and work location, age, household and personal income, and ethnicity data.

L.2.2 SCAG Year 2000 Post-Census Regional Household Travel Survey Data

The second set of data the staff evaluated was the Post-Census Regional Household Travel Survey sponsored by the Southern California Association of Governments (SCAG). SCAG is the federally designated metropolitan planning organization (MPO) for the Los Angeles region of California. The survey targeted households in the six counties of the SCAG region: Imperial, Los Angeles, Orange, San Bernardino, Riverside, and Ventura (SCAG, 2003).

SCAG survey has data of time spent at home, trip data, geo code for locations, home address, age, income, ethnicity, and limited residency duration (months lived at home location).

L.2.3 Caltrans 2000-2001 California Statewide Household Travel Survey Data

Caltrans maintains statewide household travel data to estimate, model, and forecast travel throughout the State. The information is used to help in transportation planning, project development, air quality analysis, and other programs. The CHTS obtained sample household socioeconomic and travel data at the regional and statewide levels.

In the raw survey database obtained from Caltrans, there is data about trip duration, activity duration, location type, geo-code for destination, address, age, income, and ethnicity. There is no data about residency duration.

Caltrans is currently developing a new 2011-2012 CHTS, which is a joint effort among Caltrans, SCAG and other MPOs. ARB is part of the Steering Committee.

L.2.4 Data Sources Summary

Table L.1 summarizes the activity data sources the staff analyzed, which include IPUMS census data, SCAG 2000 regional travel survey data, and Caltrans 2000-2001 CHTS data. It shows the data availability based on the HRA related categories.

Table L.1 Activity Data Sources

Sources			
HRA related Categories	IPUMS-USA Census Data 2000-2009	SCAG 2000 Travel Survey	Caltrans 2000-2001 CHTS
Residency duration	Year moved in	Months lived at home location	N/A*
Time at home per day	N/A	At home activity duration	At home activity duration
Time away from home	Hours worked, Travel time to work	Trip duration, activity duration	Trip duration, activity duration
Trip distance	N/A	Geo code for origin and destination	Geo code for destination
Residence location	City. No zip code	Address	Address
Age	Yes	Yes	Yes
Income level	Income Variables	Household income	Household income
Seasonal trend	N/A	N/A	N/A
Ethnicity	Yes	Yes	Yes
Data Set	Federal censuses (1850-2000), American Community Surveys (2000-2009)	2000-2002 Six-county Los Angeles region of CA	2000-2001 CA Statewide weekday travel survey

* N/A: Data is not available.

L.3 Methodologies and Findings:

In this section, we outline the methodologies we used in each of the data sources to estimate a person's time period lived in his residence and the time spent in different activities each day. We also examined how different environmental factors such as socioeconomic status, age, and ethnicity affect residency duration and daily activity patterns. We conclude with a discussion of the findings of each of the data sources.

L.3.1 IPUMS-USA data

L.3.1.1 Methodology

The staff used IPUMS online analysis tool (IPUMS Tool) to analyze the residency duration data based on ACS 2006-2009 data. The results are compiled and discussed below.

There are IPMUS_USA ACS data from 2000 to 2005 as well. However, the IPMUS_USA ACS data from 2006 to 2009 are more recent and have the same sample size percentage (i.e. 1%) for each year. In addition, these data include persons in group quarters and the smallest identifiable geographic unit is the Public Use Microdata Area (PUMA) containing at least 100,000 persons (IPUMS Samples). Group quarters consist of both institutions and units housing either a primary family or a primary individual plus a given number of persons unrelated to the head (IPUMA GQ).

L.3.1.2 Findings and Discussions

L.3.1.2.1 California Statewide Residency Duration Distributions

Table L.2 presents California statewide time moved into residence distributions compiled from the analysis results of ACS 2006, 2007, 2008, 2009 single year samples and ACS 2006-2008 3-year sample using IPUMS-USA online data analyzing tool. The time moved into residence variable has 7 values in ACS data as listed in “Time Moved into Residence” column in Table L.2, including “5 to 9 years ago” and “30 years ago”. The statistical data provided has the samples’ household weight applied. Household weight indicates how many households in the U.S. population are represented by a given household in an IPUMS sample (IPUMS Weights). Each cell besides the row and column headers in Table L.2 contains a household percent and the number of householders presented by that percent.

In summary, IPUMS-USA ACS 2006 to 2009 data shows that the percentage of the California householders with a residency period of 30 years or greater is less than 9%. In other words, over 91% of California householders had lived in their current residence location for less than 30 years. These data also shows that over 63% California householders had lived at their current residence for 9 years or less.

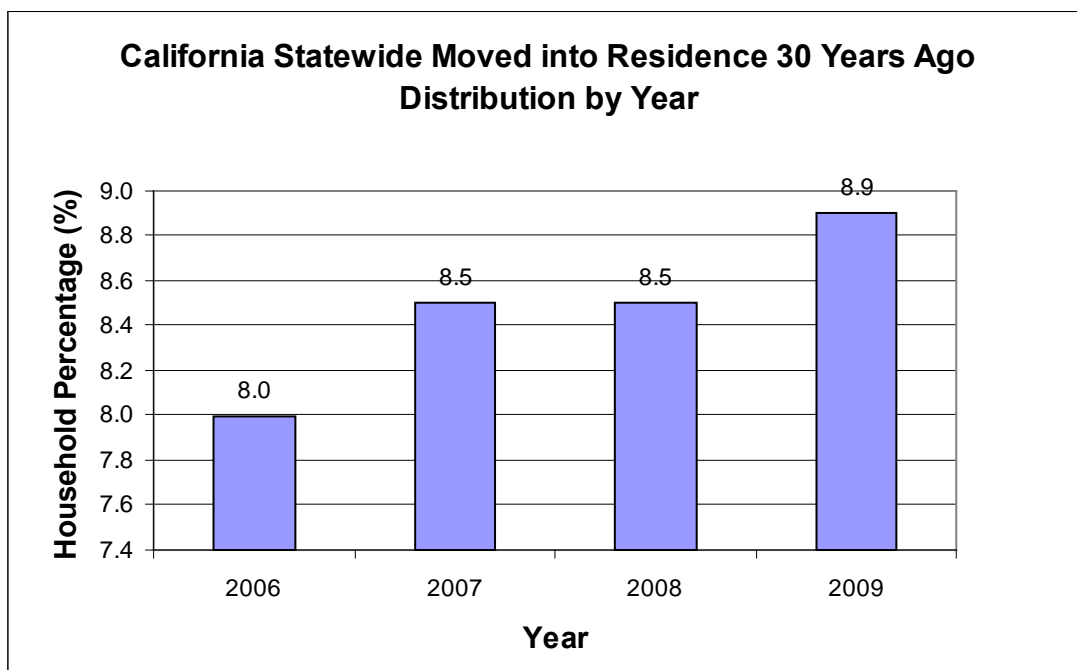
**Table L.2* California Statewide Time Moved into Residence Distribution by Year
(Weighted Household Percent and Number)**

Time Moved into Residence	2006	2007	2008	2006-2008 3-year Sample	2009
12 months or less	17.2 2,084,533.0	15.9 1,939,774.0	15.4 1,871,049.0	16.2 1,968,717.0	15.7 1923501
13 to 23 months ago	7.5 910,536.0	6.9 838,322.0	6.5 796,030.0	7 848,579.0	6.4 783261
2 to 4 years ago	21.9 2,665,547.0	22.9 2,795,422.0	23.3 2,834,921.0	22.7 2,768,053.0	20.3 2482340
5 to 9 years ago	19.8 2,411,057.0	20.1 2,449,371.0	20.1 2,448,160.0	20 2,434,099.0	20.9 2554979
10 to 19 years ago	17.6 2,141,482.0	17.7 2,162,519.0	18.1 2,208,805.0	17.8 2,169,353.0	18.9 2311981
20 to 29 years ago	7.9 960,926.0	8.1 982,699.0	8.0 979,208.0	8.0 974,196.0	8.7 1067833
30 years ago	8.0 977,136.0	8.5 1,032,572.0	8.5 1,038,566.0	8.3 1,014,849.0	8.9 1090992
TOTAL	100.0 12,151,217.0	100.0 12,200,679.0	100.0 12,176,739.0	100.0 12,177,846.0	100.0 12214887

* IPUMS-USA ACS 2006 to 2009 data with household weight applied. As of March 2011, there is no IPUMS-USA multi-year sample with ACS 2009 sample included yet.

Figure L.1 graphically depicts the 2006 to 2009 statewide householder percentages of Californians that moved into their current home location 30 years ago. From 2006 to 2009, this figure shows an increase in the percentage of statewide householders that moved into residence 30 years ago.

Figure L.1*

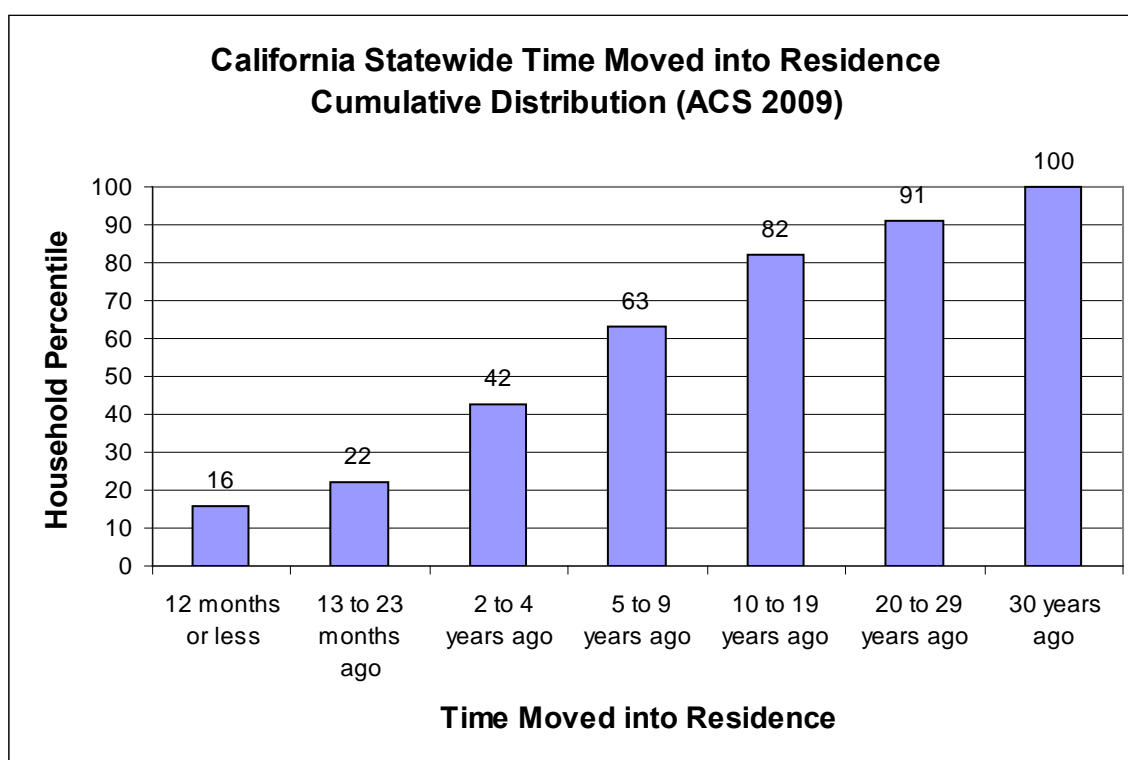


* IPUMS-USA ACS 2006, 2007, 2008, and 2009 single year samples with household weight applied.

Figure L.2 and Figure L.3, respectively, show the California statewide time moved into residence cumulative distributions using IPUMS-USA ACS 2009 sample and 2006-2008 3-year sample with household weight applied. Both of these figures show that over 90 percent of California householders had lived at their current home address for less than 30 years; and approximately 63 to 66 percent of the householders had lived at their current residency location for 9 years or less.

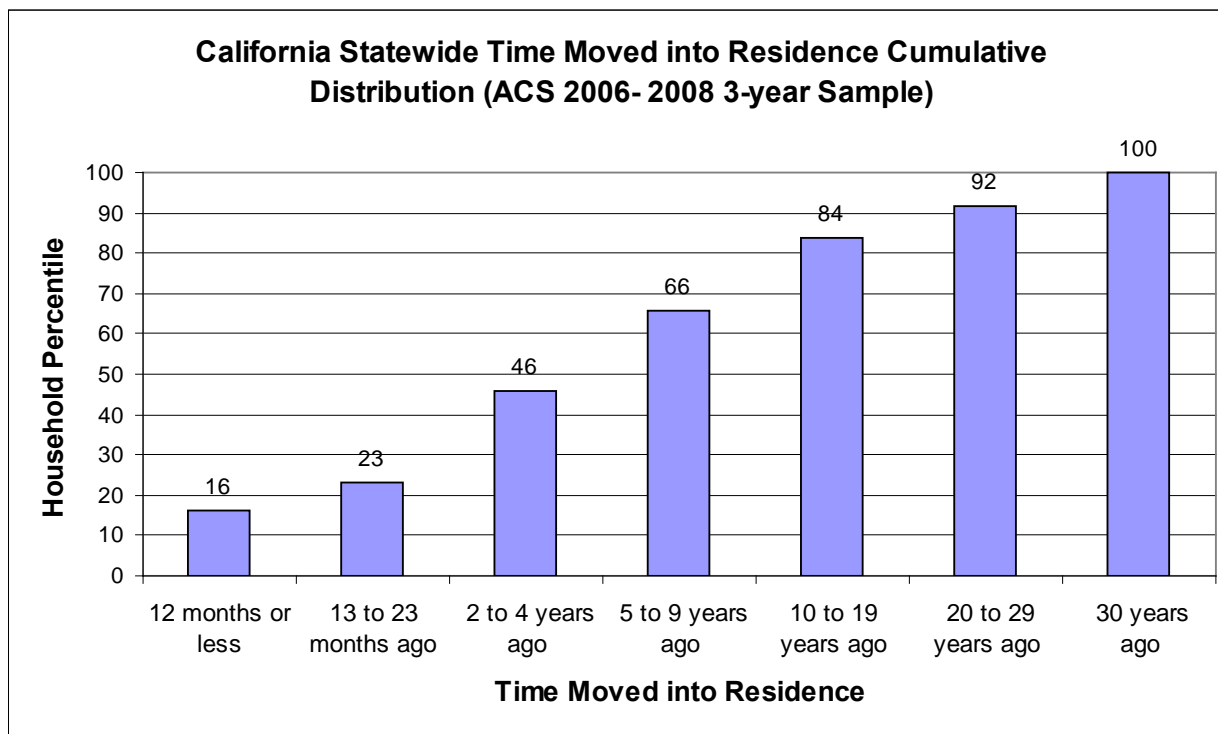
See Supplemental Information section (page 28) for additional information on time moved into residence distributions by California householder's ethnicity, age, and household income from IPUMS-USA ACS 2009 data.

Figure L.2*



* IPUMS-USA ACS 2009 data with household weight applied.

Figure L.3*



* IPUMS-USA ACS 2006-2008 3-year sample with household weight applied. As of March 2011, there is no IPUMS-USA multi-year sample with ACS 2009 sample included available yet.

L.3.1.2.2 Evaluation of Populations and Residency Duration Distributions for California Cities

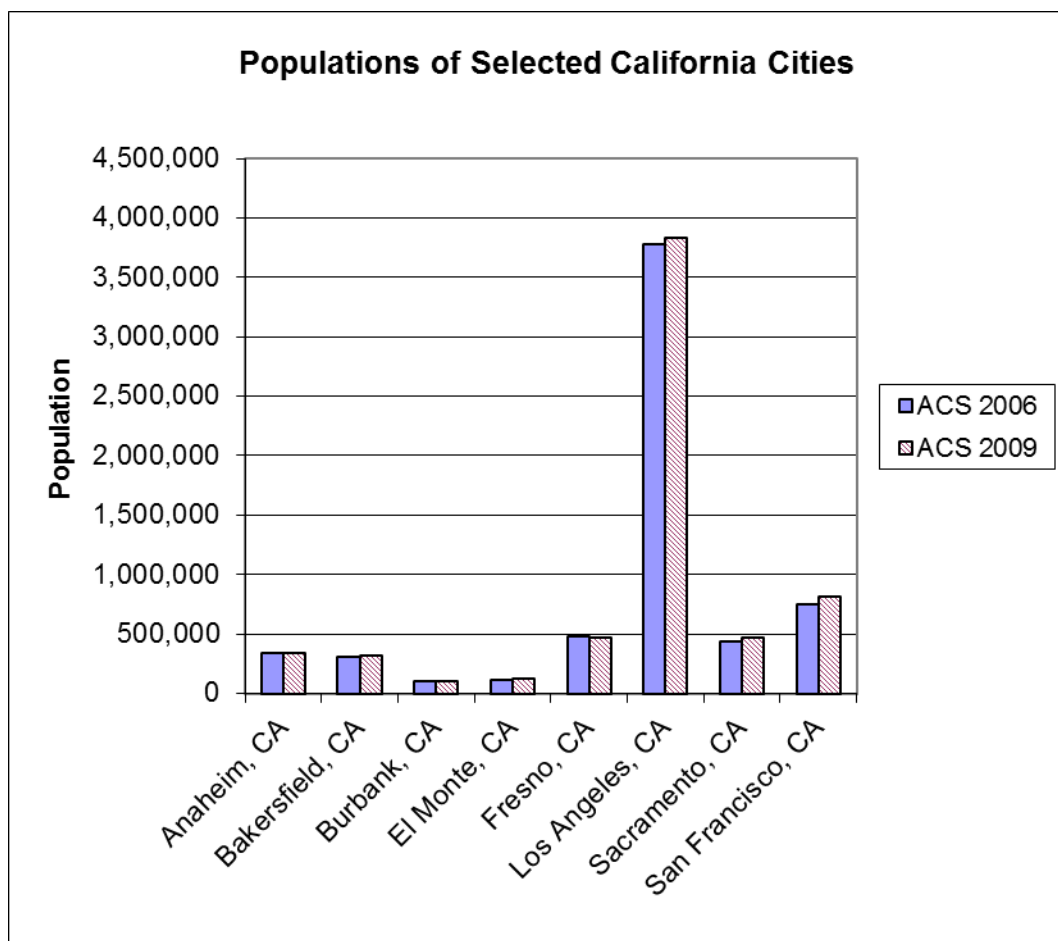
Table L.3 and Figure L.4 display the populations and population changes for 8 selected California cities from IPUMS-USA ACS 2006 to ACS 2009 data with person weight applied. Person weight indicates how many persons in the U.S. population are represented by a given person in an IPUMS sample (IPUMS Weights). These 8 cities have populations over 100,000 from IPUMS-USA ACS 2006 and 2009 data, and were selected to represent the regions of California and to include an Environmental Justice community (Fresno, CA). If an area consisted of less than 100,000 persons then it was combined with another area so that the total population would be greater than 100,000 persons. The exhaustive distribution data from IPUMS-USA ACS 2006 and 2009 samples contain 41 identifiable California cities.

Table L.3* Comparison of Populations of Selected California Cities (IPUMS-USA ACS 2006 and 2009)

California City	Anaheim, CA	Bakersfield, CA	Burbank, CA	El Monte, CA	Fresno, CA	Los Angeles, CA	Sacramento, CA	San Francisco, CA
2006	343,120	304,813	107,540	113,644	474,466	3,775,106	438,385	744,389
2009	337,966	316,313	103,096	121,183	466,466	3,832,554	466,492	815,575
Population Change Percent	-1.5	3.8	-4.1	6.6	-1.7	1.5	6.4	9.6

* IPUMS-USA ACS 2006 and 2009 data with person weight applied.

Figure L.4*



* IPUMS-USA ACS 2006 and 2009 data with person weight applied.

Table L.4 and L.5 display the time moved into residence distributions for the 8 selected California cities from IPUMS-USA ACS 2006 and 2009 data, respectively, with household weight applied. Both tables show that 89% to 96% of householders had moved out of their residence within 30 years. In other words, about 4% to 11% householders had lived at their current residence for 30 years or longer. The residency duration data from IPUMS-USA ACS also indicates that, for all the 41 identifiable California cities, there are about 1% to 15% of householders had lived at their current residence for 30 years or longer in 2006, whereas 2% to 15% of householders had lived at their current residence for 30 years or longer in 2009.

Table L.4* Time Moved into Residence Distribution for Selected California Cities Weighted Household Percent and Samples (IPUMS-USA ACS 2006)

Time Moved into Residence	Anaheim, CA	Bakersfield, CA	Burbank, CA	El Monte, CA	Fresno, CA	Los Angeles, CA	Sacramento, CA	San Francisco, CA
12 months or less	19.1 18,845	23.6 23,729	11.3 4,847	11 3,083	22 33,457	15.8 200,769	21.9 37,111	15.8 50,869
13 to 23 months ago	8.1 8,021	9.1 9,194	9.9 4,236	6.1 1,715	7.2 10,896	6.4 81,792	9.3 15,778	7.9 25,535
2 to 4 years ago	22.9 22,542	25.9 26,028	21.8 9,314	23 6,456	24.3 36,928	21.8 278,034	23.2 39,271	21 67,837
5 to 9 years ago	21.6 21,324	18.9 19,038	23.2 9,924	23.1 6,469	19.8 30,086	22.3 284,354	17.7 30,006	15.6 50,166
10 to 19 years ago	15.6 15,341	13.3 13,427	15.5 6,649	18.4 5,177	14.9 22,728	18.1 231,199	11.2 18,986	20.2 65,170
20 to 29 years ago	4.9 4,838	5.3 5,373	7.5 3,194	9.9 2,768	5.6 8,512	7.3 93,569	7.8 13,134	9 28,989
30 years ago	7.8 7,654	3.8 3,857	10.9 4,651	8.5 2,397	6.3 9,554	8.2 104,450	8.8 14,939	10.5 33,980
TOTAL	100 98,565	100 100,646	100 42,815	100 28,065	100 152,161	100 1,274,167	100 169,225	100 322,546

* IPUMS-USA ACS 2006 data with household weight applied.

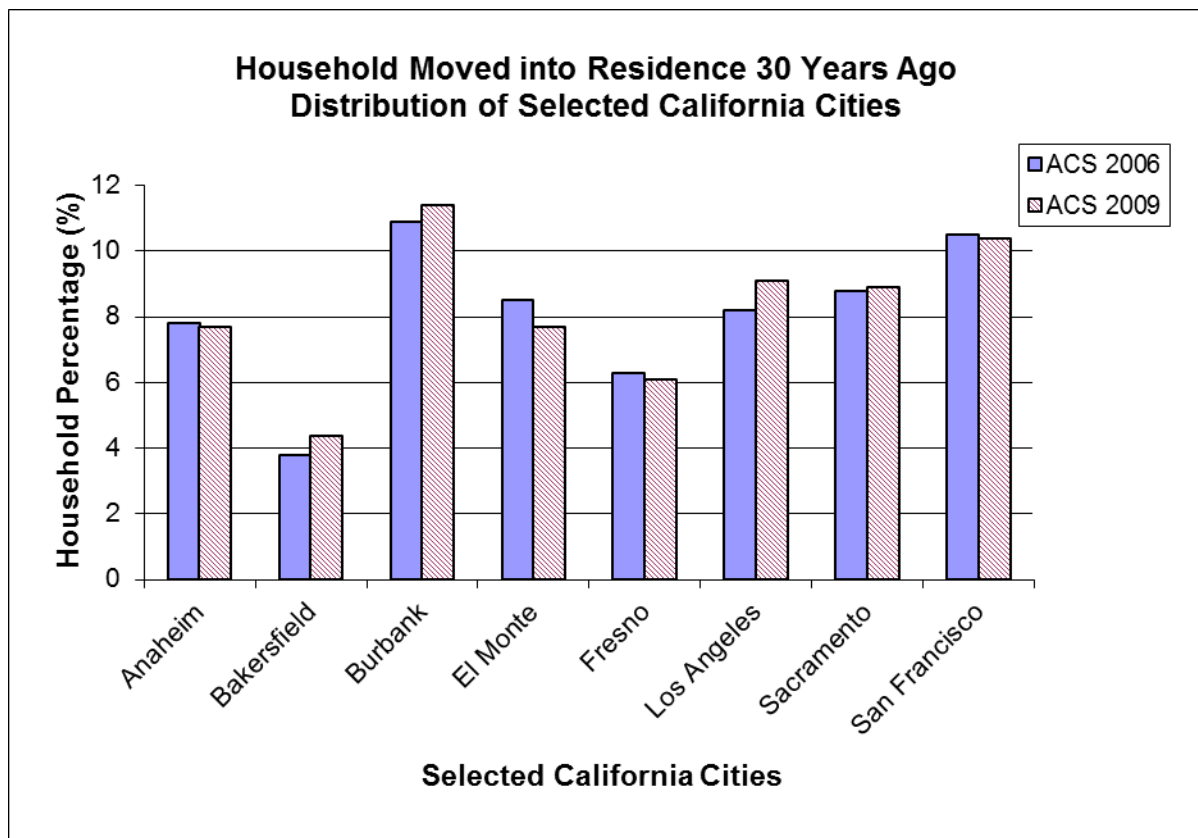
**Table L.5* Time Moved into Residence Distribution for Selected California Cities
Weighted Household Percent and Samples
(IPUMS-USA ACS 2009)**

Time Moved into Residence	Anaheim, CA	Bakersfield, CA	Burbank, CA	El Monte, CA	Fresno, CA	Los Angeles, CA	Sacramento, CA	San Francisco, CA
12 months or less	15.8 15,554	21.3 21,302	17.5 6,907	11 2,995	21.3 31,605	15.5 200,860	23 40,825	14.8 48,036
13 to 23 months ago	6.5 6,428	7.9 7,875	6.3 2,475	6.9 1,888	8.8 13,032	5.7 74,089	8.4 14,879	7 22,627
2 to 4 years ago	22.7 22,405	27.1 27,146	19.2 7,580	19.7 5,388	19.8 29,474	20.3 263,922	22.3 39,562	21.9 71,210
5 to 9 years ago	21.1 20,817	20.4 20,411	21.5 8,507	26.8 7,337	20.2 29,998	21.6 279,991	17.4 30,875	18.7 60,640
10 to 19 years ago	19.2 18,951	14.6 14,640	18.7 7,391	17.2 4,692	16.9 25,153	20.2 262,938	13.2 23,382	18.6 60,314
20 to 29 years ago	7.1 6,964	4.2 4,241	5.5 2,170	10.7 2,932	6.9 10,258	7.6 98,225	6.7 11,848	8.7 28,132
30 years ago	7.7 7,591	4.4 4,443	11.4 4,504	7.7 2,094	6.1 8,989	9.1 118,599	8.9 15,830	10.4 33,631
TOTAL	100 98,710	100 100,058	100 39,534	100 27,326	100 148,509	100 1,298,624	100 177,201	100 324,590

* IPUMS-USA ACS 2009 data with household weight applied.

Figure L.5 shows the distribution of householders with residency periods of 30 years or greater for the 8 selected California cities from IPUMS-USA ACS 2006 to ACS 2009 data with household weight applied.

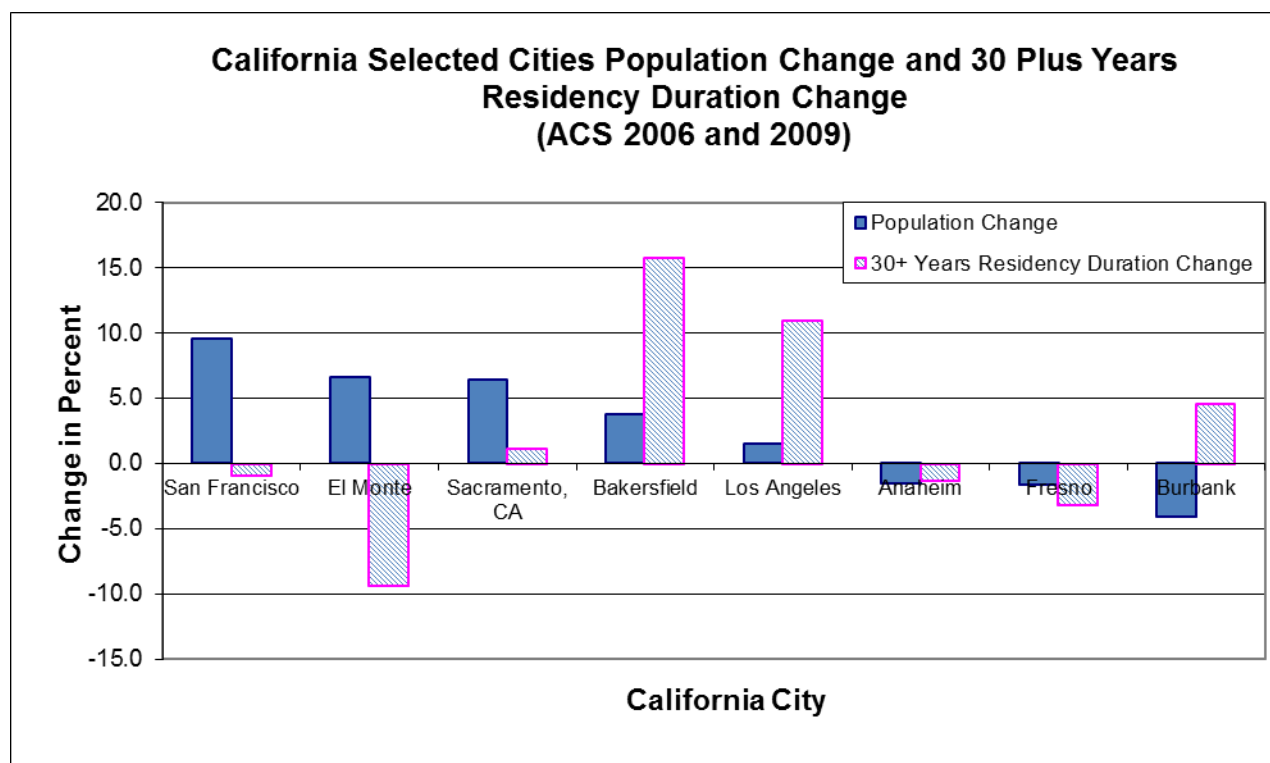
Figure L.5*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied.

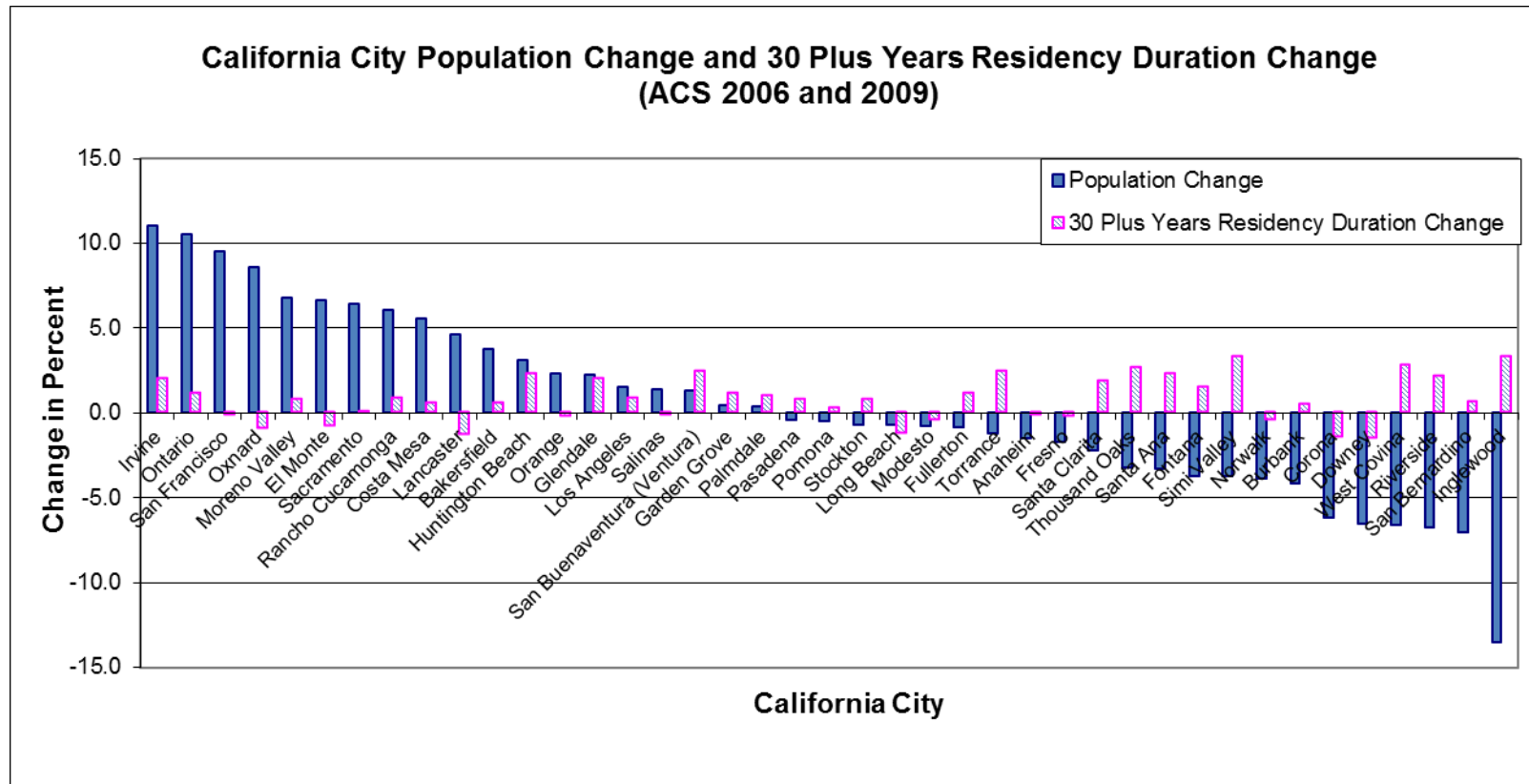
Staff also analyzed the population changes and the 30 years or greater residency duration changes for both the 8 selected cities and the 41 identifiable California cities using IPUMS-USA ACS 2006 to ACS 2009 data. The purpose of this analysis is to see if a rapidly growing city has a different pattern of residency durations. The results are illustrated in Figure L.6 and Figure L.7 respectively. There is no obvious correlation found between the population changes and the 30 years or greater residency duration changes. Figure L.7 shows that, when the population increased from 2006 to 2009, there were 13 cities showed an increase in 30 years or greater residency duration while 6 cities showed a decrease in 30 years or greater residency duration. And when the population decreased from 2006 to 2009, there were 15 cities showed an increase in 30 years or greater residency duration while 7 cities showed a decrease in 30 years or greater residency duration.

Figure L.6*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied to the residency duration data, and person weight applied to population data.

Figure L.7*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied to the residency duration data, and person weight applied to population data.

L.3.1.3 Limitations of the IPUMS-USA data for Our Purposes

The ideal data for our purposes would be longitudinal data on the duration of residence of individuals. The IPUMS data collects information on how long the person has been in the current residence, but not previous residences. People may continue at the current residence for an indefinite period of time. Likewise people who report living in the current residence for a short period of time may have lived in the previous residence for an extended period time. This could be the case with older people who have recently moved to assisted living. Data on the amount of time that a person might have lived beyond thirty years were not collected. There is therefore no way of knowing the number of people who may have lived in the same residence for 40 or 50 years. Geographic areas with fewer than 100,000 inhabitants are not identifiable so the impact of living in a smaller community on residency time in California could not be determined. The data are binned into intervals that are as much 9 years at the longer residency times. These data are the only California specific data that we could locate however, and are generally supportive of the nationwide data. .

L.3.2 SCAG Year 2000 Post-Census Regional Household Travel Survey Data

L.3.2.1 Methodology

The survey collected demographic information about persons and households. It also captured activity and travel information for household members during a 24-hour or 48-hour timeframe. The survey coincides with 2000-2001 CHTS. According to the 2000 Census, this region had 5,386,491 households. The total number of households that participated the survey and met the criteria for a completed record was 16,939 (SCAG, 2003). In the survey report, there are some trip time and age information.

Using the SCAG survey database, a statistical analysis for the regional average time spent at home per day was performed.

L.3.2.2 Findings and Discussions

The average time at home per person per day was determined to be 17.6 hours, which is about 73% of a day. This result is based on 44,344 person day records without any weight factor applied.

The residency duration data (months lived at home location) in the database is labeled as 1-12, 98-unknown, and 99-refused. Label 1-11 represent 1 to 11 months lived at home location, whereas label 12 represents 12 plus months lived at home location. No additional data was collected on residency duration. Therefore, the residency duration data from SCAG survey is limited for long-term health risk assessment evaluations.

L.3.2.3 Limitations on the Use of SCAG Household Travel Survey Data

The limitations of SCAG travel survey data include that the time spent at home analysis does not have weight factors applied due to in-sufficient user information on weights for personal level analysis (SCAG Manual); and the residency duration is not further categorized for period that is 12 months or longer, which limits the data usage for long-term health risk assessment.

L.3.3 Caltrans 2000-2001 California Statewide Household Travel Survey Data

L.3.3.1 Methodology

The Survey was “activity” based and included in-home activities and any travel to activity locations. The Survey was conducted among households in each of the 58 counties throughout the State and grouped by region to provide a snapshot of both regional and interregional travel patterns. The participating households were asked to record travel information in their diaries for a specified 24-hour or 48-hour period. The Survey produced a sample size of 17,040 randomly selected households with an overall standard error of 0.8% at the 95% confidence level with respect to household level attributes at the statewide level of analysis (CHTS, 2003).

There are statistical survey reports about income, region, trip purpose, and trip time (home-work travel time percent by five minutes intervals by region). However, no report is based on travel distance, activity duration, season, or weekend.

A statistical analysis was performed by the staff using the CHTS database for the statewide average time spent at home per person per day. The result is based on 40,696 person day respondents’ records without any population weight factor applied.

Further statistical analysis gave us the statewide time at home average by age group, income level, and ethnicity. Time at home by age group and ethnicity results are based on 40,653 person day records. Time at home by income level result is based on 40,696 person day records. These results don’t have any weight factors applied. And five percent of the person day records are weekend records.

L.3.3.2 Findings and Discussions

L.3.3.2.1 California Statewide Average Time Spent at Home and Distributions by Age, Income, and Ethnicity

The statewide average time spent at home per person per day was determined to be 17.5 hours (including weekend samples), which is 73% of a day. This statewide average time at home percentage is about the same as the SCAG’s regional average time at home percentage based on its 2000 regional travel survey data.

Table L.6 and Figure L.8 demonstrate California statewide time spent at home distribution by age group. The results show that children less than 2 years old spend

85% of their time at home, which is 12% more than the statewide average 73%. Children in the age group 2 to <16 spend 72% of their time at home, which is a little less than the statewide average time at home.

Age groups listed in Table L.6 match those used for the application of Age Specific Sensitivity Factors that are listed in OEHHA's Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing or available values, and adjustments to allow for early life stage exposures (May 2009).

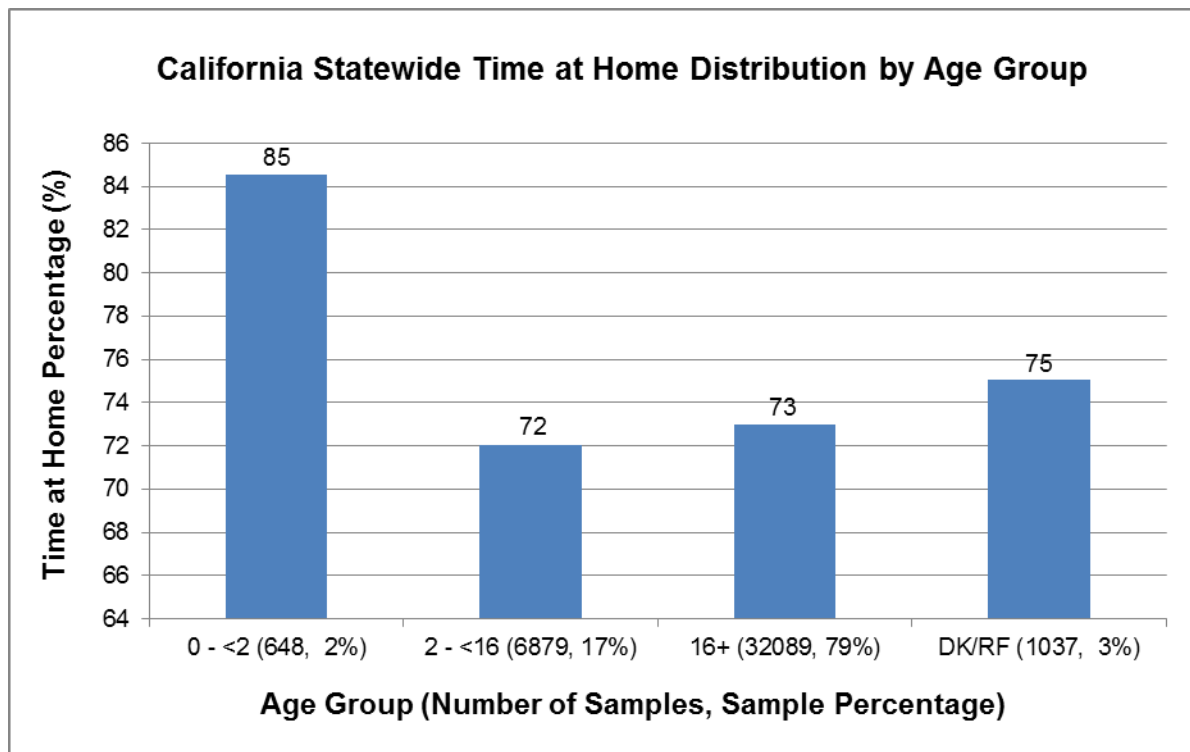
Table L.6 California Statewide Time at Home Distribution by Age Group

Age Group	Time at Home in Minute	Time at Home in Hour	Time at Home Percentage	Number of Samples	Sample Percentage
0 - <2	1218	20.3	85	648	2%
2 - <16	1037	17.3	72	6879	17%
16+	1051	17.5	73	32089	79%
DK/RF	1081	18.0	75	1037	3%
State Avg.	1052	17.5	73	40653	100%

Notes:

1. Caltrans 2000-2001 CHTS Data.
2. DK/RF means Don't Know/Refused.
3. Results don't have any weight factors applied.

Figure L.8



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. DK/RF means Don't Know/Refused.
3. California statewide time at home average is 73%.
4. Total number of samples: 40,653.
5. Results don't have any weight factors applied.

Table L.7 and Figure L.9 demonstrate California statewide time spent at home distribution by household income level. They show a trend as the higher the household income is, the less time people spend at their home. The households with income level less than \$10k spend most of their time at home as 81% (19.5 hr.) whereas the households with income level more than \$100k but less than \$150k spend the least time at home as 68% (16.2hr). The households with income level more than \$35k but less than \$50k spend the state average time 73% (17.5hr) at home.

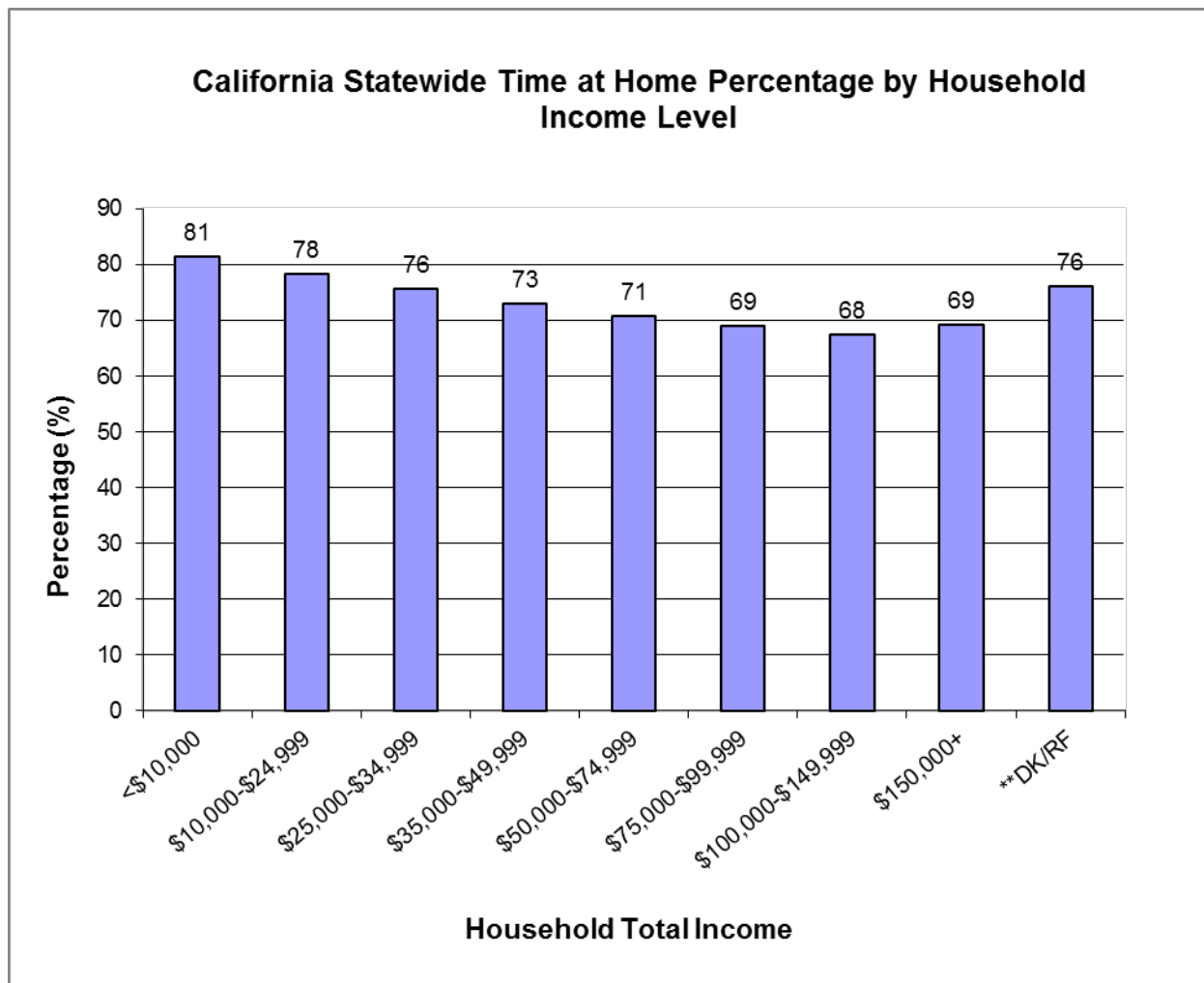
Table L.7 California Statewide Time at Home Distribution by Household Income Level

Household Total Income	Time at Home In Minute	Time at Home In Hour	Time at Home Percentage	Number of Samples	Sample Percentage
<\$10,000	1172	19.5	81	1312	3%
\$10,000-\$24,999	1128	18.8	78	5189	13%
\$25,000-\$34,999	1089	18.2	76	5265	13%
\$35,000-\$49,999	1051	17.5	73	5568	14%
\$50,000-\$74,999	1019	17.0	71	8677	21%
\$75,000-\$99,999	994	16.6	69	5077	12%
\$100,000-\$149,999	973	16.2	68	3332	8%
\$150,000+	998	16.6	69	1525	4%
DK/RF	1095	18.3	76	4751	12%
Total				40696	100%

Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Results don't have any weight factors applied.

Figure L.9



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Total number of samples: 40,696.
5. Results don't have any weight factors applied.

Table L.8 and Figure L.10 show California statewide time spent at home distribution by ethnicity. They depict that all the ethnic groups spend 71% to 74% time at home per day. The N/A in the ethnicity group in Table L.8 means the description of the ethnicity code 6 in the database is not available. The Caltrans survey data contact person believes that the code 6 should not have existed. This data was a mistake in survey reporting. The 532 person day records (1% of the total person day records) with ethnicity code 6 may exist in error.

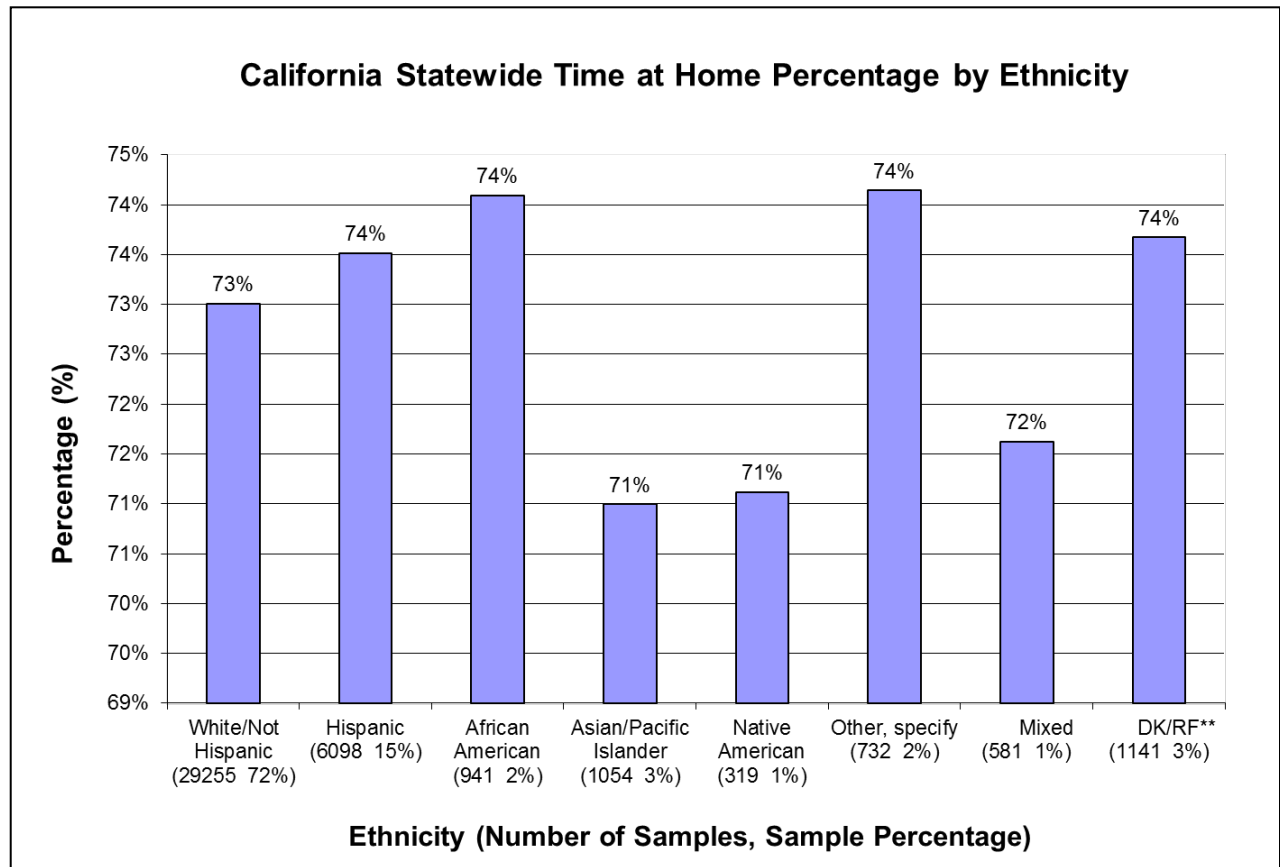
Table L.8 California Statewide Time at Home Average by Ethnicity

Ethnicity	Ethnicity Code	Time at Home In Minute	Time at Home In Hour	Time at Home Percentage	Number of Samples	Sample Percentage
White/Not Hispanic	1	1051	17.5	73%	29255	72%
Hispanic	2	1059	17.6	74%	6098	15%
African American	3	1067	17.8	74%	941	2%
Asian/Pacific Islander	4	1022	17.0	71%	1054	3%
Native American	5	1024	17.1	71%	319	1%
N/A	6	1077	17.9	75%	532	1%
Other, specify	7	1068	17.8	74%	732	2%
Mixed	8	1031	17.2	72%	581	1%
DK/RF	9	1061	17.7	74%	1141	3%
Total					40653	100%

Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. N/A means the description of ethnicity code 6 is not available.
5. Results don't have any weight factors applied.

Figure L.10



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Total number of samples: 40,653.
5. Results don't have any weight factors applied.

L.3.3.2.2 Comparison of Time at Home Results from CHTS Data with Time inside Home Results from ARB Activity Pattern Studies

Staff compared the time at home by age group statistical results from Caltrans 2000-2001 CHTS data and the time inside home results from 1987-1990 ARB activity pattern studies (ARB, 2005). Table L.9 and Figure L.11 show that, compare to the time spent inside home in 1987-1990, children under age of 12 spent similar amount of time at home in 2000-2001. However, teens (age 12 to 17) spent 6% more time at home in 2000-2001, and adults spent 11% more time at home in 2000-2001.

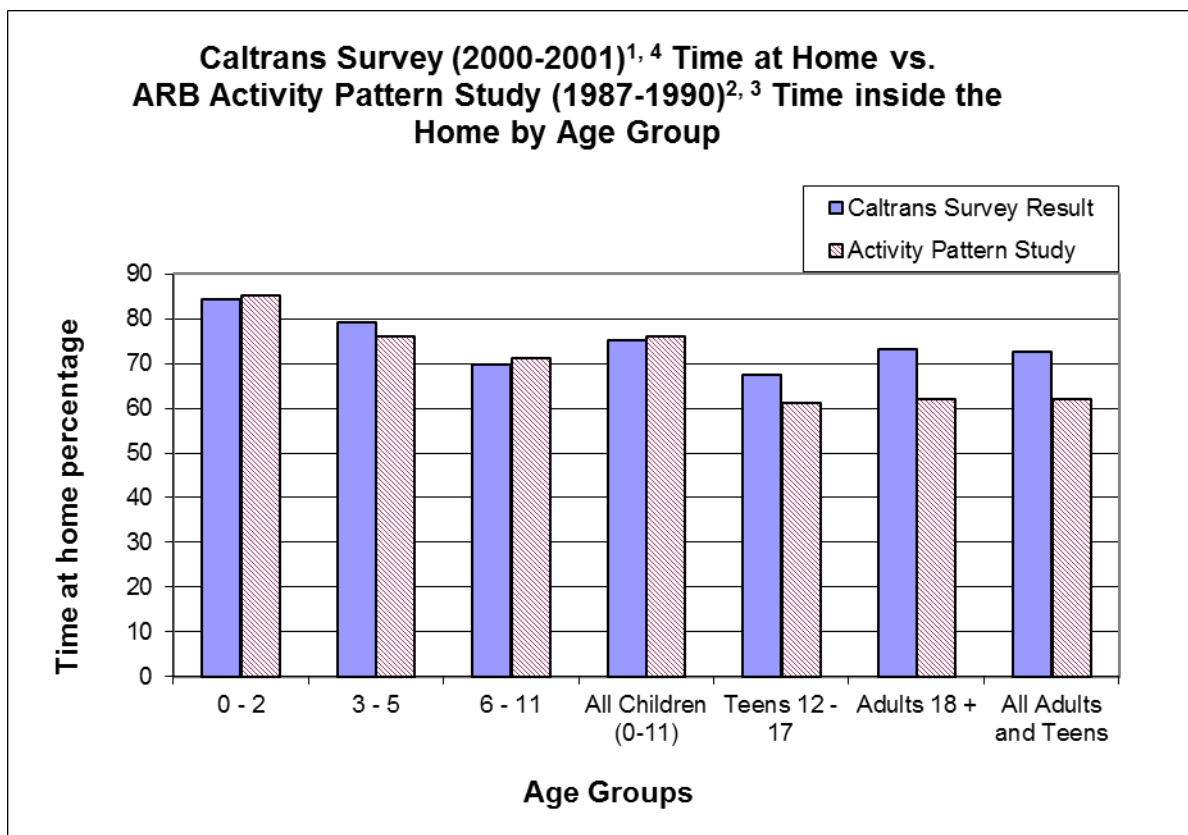
Table L.9 Caltrans Survey (2000-2001) Time at Home vs. ARB Activity Pattern Study (1987-1990) Time inside the Home by Age Group

Age Group	Caltrans ^{1, 4}			ARB ^{2, 3}	
	Number of Samples	Time at Home In Hour	Time at Home (%)	Number of Samples	Time Inside Home (%)
0 - 2	1086	20.3	84	313	85
3 - 5	1328	19.0	79	302	76
6 - 11	2985	16.8	70	585	71
All Children (0-11)	5399	18.0	75	1200	76
Teens 12 - 17	3180	16.2	67	183	61
Adults 18 +	31937	17.6	73	1579	62
All Adults and Teens	34217	17.4	73	1762	62

Notes:

1. The 2000 - 2001 California Statewide Household Travel Survey was conducted among households in each of the 58 counties throughout the State and grouped by region. Total person day records are 40,653.
2. The 1989 -1990 Children's Activity Pattern Study's samples are selected from households among three major areas: Southern Coast, S.F. Bay Area, and the rest of state. Total samples are 1,200 (ARB, 1991).
3. The 1987 – 1988 California Residents Activity Pattern Study's samples are selected from the same three major areas as for Children's Activity Pattern Study, with 1579 adult samples and 183 youth samples (ARB, 1992).
4. Results from Caltrans survey data don't have any weight factors applied, whereas the results from the activity pattern studies have the weight factors applied.

Figure L.11



Notes:

1. The 2000 - 2001 California Statewide Household Travel Survey was conducted among households in each of the 58 counties throughout the State and grouped by region. Total person day records are 40,653.
2. The 1989 -1990 Children's Activity Pattern Study's samples are selected from households among three major areas: Southern Coast, S.F. Bay Area, and the rest of state. Total samples are 1,200 (ARB, 1991).
3. The 1987 – 1988 California Residents Activity Pattern Study's samples are selected from the same three major areas as for Children's Activity Pattern Study, with 1579 adult samples and 183 youth samples (ARB, 1992).
4. Results from Caltrans survey data don't have any weight factors applied, whereas the results from the activity pattern studies have the weight factors applied.

L.3.3.3 Limitations on the Use of 2000-2001 CHTS data

The limitations of the use of the 2000-2001 CHTS data are that the analysis results do not have weight factors applied due to in-sufficient user information on weights for personal level analysis (CHTS Guide). And 2000-2001 CHTS does not have residence duration data.

L.4 Other Data Sources Not Used in This Report

L.4.1 The 2009 National Household Travel Survey

The 2009 NHTS updates information gathered in the 2001 NHTS and in prior Nationwide Personal Transportation Surveys. The data is collected on daily trips taken in a 24-hour period (NHTS, 2009). Although we may be able to analyze the 2009 NHTS data to get the time at home statistical results for Californians, the staff didn't use the data because the user manual was not ready at the time the staff was preparing this report.

L.4.2 National Human Activity Pattern Survey

NHAPS was sponsored by the U.S. Environmental Protection Agency. It was conducted between late September 1992 and September 1994, which collected 24-hour activity diaries and answers of personal and exposure questions. The survey interviewed 9386 participants across the 48 contiguous states (Klepeis et al., 1995).

NHAPS has time in a residence data from California respondents. However, the staff didn't further analyze this data because the 2000-2001 CHTS provides much larger sample size and more recent California-specific data.

L.5 Conclusion

The staff has evaluated several data sources to identify the California statewide exposure duration and exposure frequency characteristics. Estimates on residence duration and time spent at home have been determined from available data on the California population. The data on residency time is similar to the available national data as discussed in Chapter 11. There is some variability on the residence duration and time spent at home by ethnicity, age, and income.

The IPUMS-USA census data shows that, from 2006 to 2009, over 90% of California householders had lived at their current home address for less than 30 years, and over 63% householders had lived at their current residence for 9 years or less.

The 2000-2001 CHTS data shows that, on average, Californians spend approximately 73% of their time at home per day. When looking at the data by age group, the time increases to 85% for children under 2 years old. Children that are 2 years or older but less than 16 years old spend 72% of their time at home; whereas Californians that are 16 years or older spend 73% of their time at home. In addition, All ethnicity groups spend 71%-74% of their time at home. The data also demonstrates a trend that the higher the total household income is, the less time the residents spend at their home.

These data are the best available on the California population for helping to come up with default recommendations for the Hot Spots program.

L.6 References

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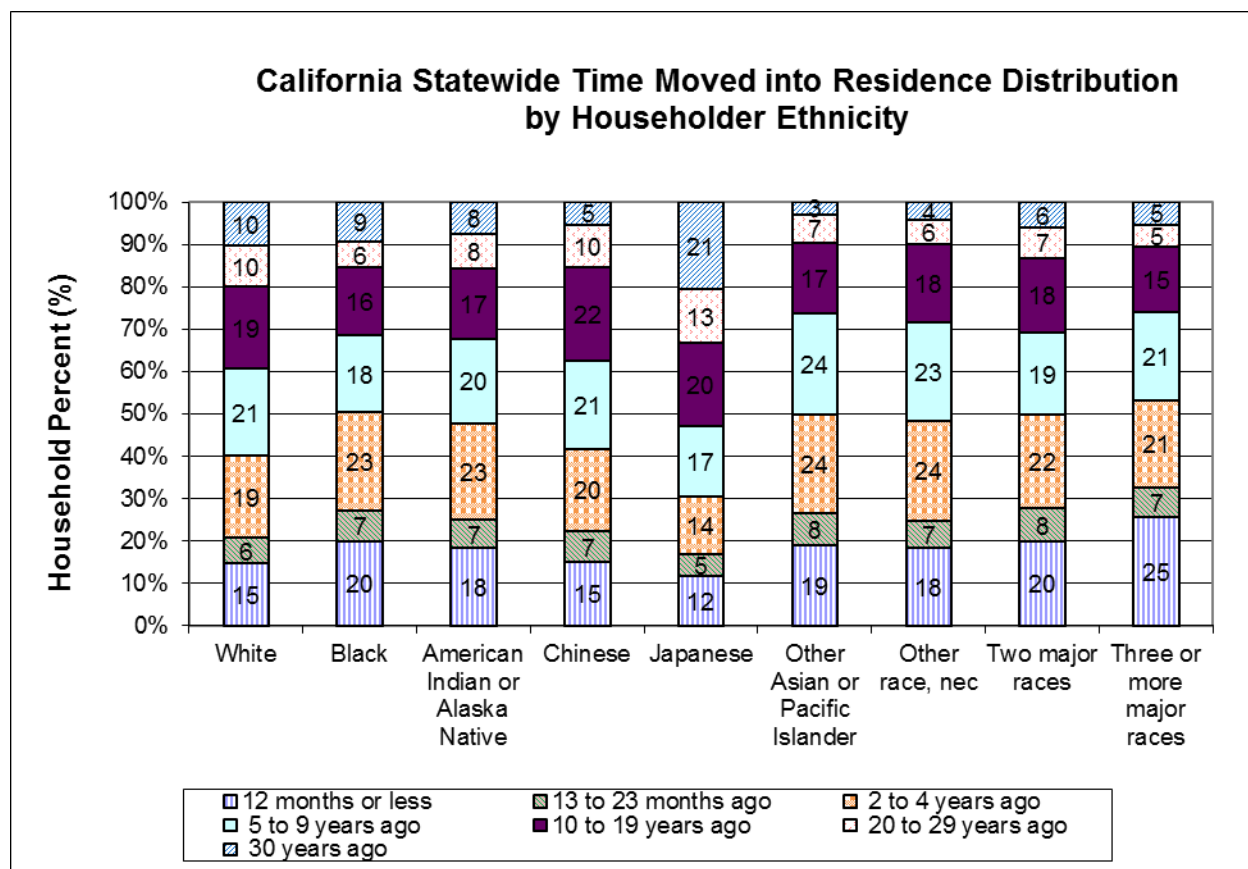
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<http://nhts.ornl.gov/introduction.shtml>. Last visited: January, 2011.
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A. Supplemental Information

The following figures graphically present the analysis results of California statewide time moved into residence distribution by householders' ethnicity, age, and household income respectively from IPUMS-USA ACS 2009 data (IPUMS-USA). The data is obtained by using IPUMS online analysis tool (IPUMS Tool). These data may be useful to the risk manager in considering population risk in different communities.

Figure A.1 shows California statewide time moved into residence distribution by householders' ethnicity. In general, the percentages of householders that moved into their residence 12 months or less ago, 2 to 4 years ago, 5 to 9 years ago, and 10 to 19 years ago are larger than the percentages of 13 to 23 months ago, 20 to 29 years ago, and 30 years ago.

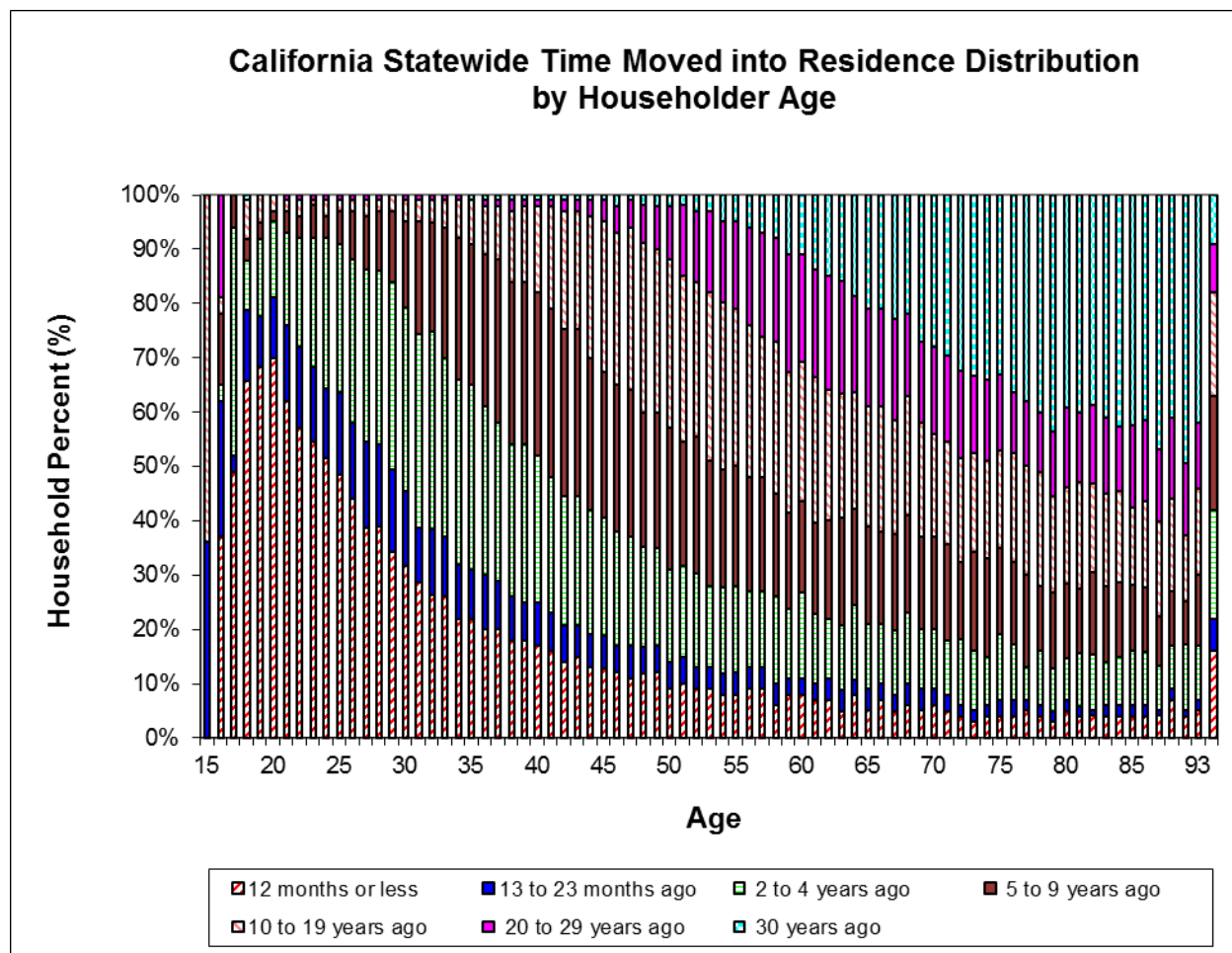
Figure A.1*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights) (IPUMS Ethnicity).

Figure A.2 presents California statewide time moved into residence distribution by householders' age. It shows a general trend that the younger the householders are, the more householders moved into their residence within the last 12 months. And the older the householders are, the more householders moved into their residence 30 years ago. There are some exceptions at the both ends of the age range.

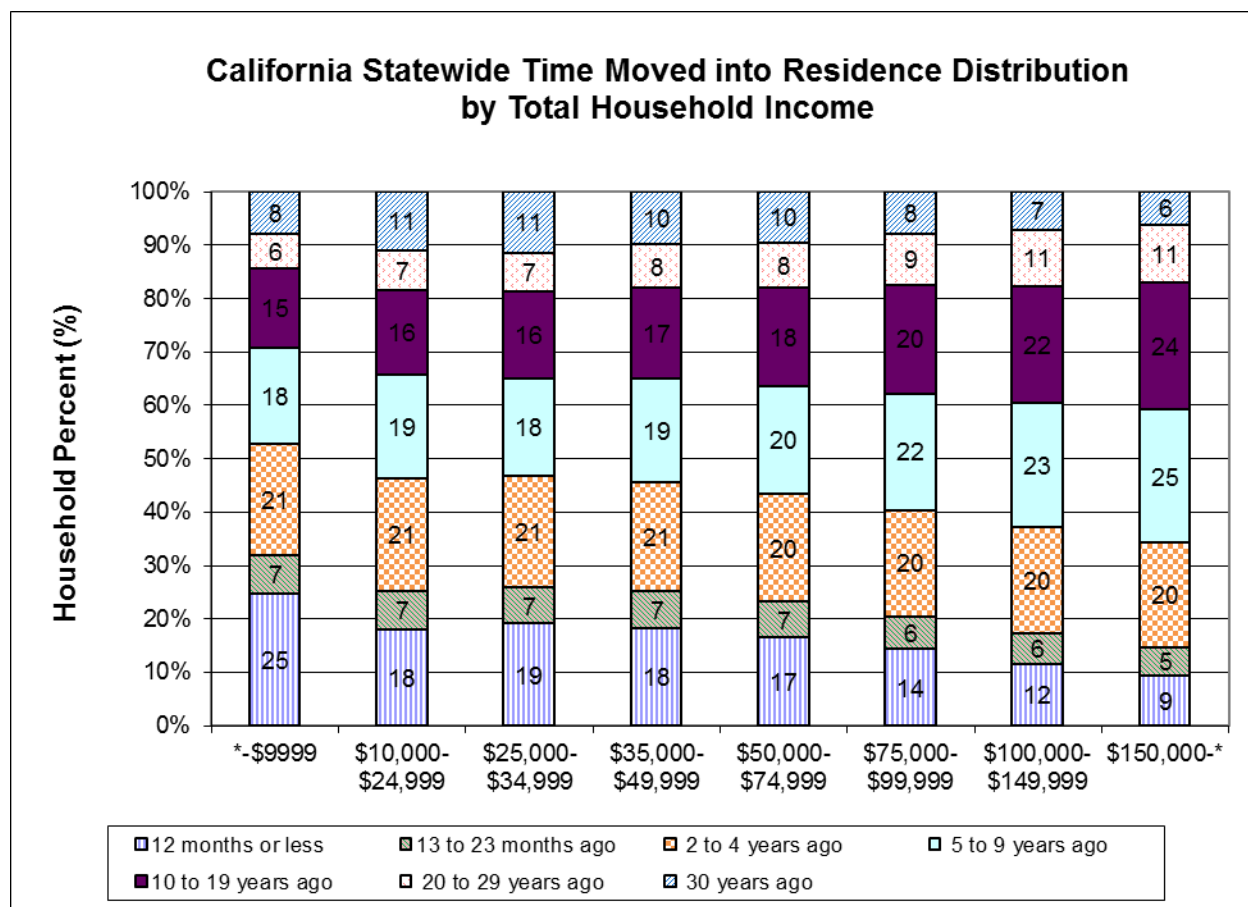
Figure A.2*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights). The age categories are 15-89 and 93.

Figure A.3 shows California statewide time moved into residence distribution by total household income. It reveals a general trend that the higher the household income is, the smaller percentage of the householders moved into their residence within last 12 months. And the households with household income of \$150,000 or above not only have the smallest percentage of householders moved into their residence within last 12 months, but also have the smallest percentage of householders moved into their residence 30 years ago.

Figure A.3*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights).

A. References

1. (IPUMS-USA) Steven Ruggles, J. Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B. Schroeder, and Matthew Sobek. *Integrated Public Use Microdata Series: Version 5.0* [Machine-readable database]. Minneapolis: University of Minnesota, 2010. <http://usa.ipums.org/usa/index.shtml>. Last visited: January, 2011.
2. (IPUMS Tool) IPUMS Online Data Analysis System: <http://usa.ipums.org/usa/sda/>. Last visited: January, 2011.
3. (IPUMS Ethnicity) IPUMS-USA Ethnicity Code: <http://usa.ipums.org/usa-action/codes.do?mnemonic=RACE>. Last visited: January, 2011.
4. (IPUMS Weights) IPUMS-USA Sample Weights: <http://usa.ipums.org/usa/intro.shtml#weights>. Last visited: January, 2011.

Appendix M.

How to Post-Process Offsite Worker Concentrations using the Hourly Raw Results from AERMOD

Appendix M

How to Post-Process Offsite Worker Concentrations using the Hourly Raw Results from AERMOD

The offsite worker health risk analysis begins with estimating the pollutant concentration at a receptor location. To estimate this concentration, the typical approach is to use the residential annual concentration that is modeled based on the adjacent facility's emission schedule. However, if the facility emissions are non-continuous (i.e., the facility does not emit for 24 hours a day and 7 days a week), the residential concentration may not represent what the offsite worker breathes during their work shift. In lieu of conducting additional special case modeling which can be time-consuming, the residential annual concentration is adjusted upwards using a worker adjustment factor based on the facility's emission schedule with respect to the worker's schedule. For an 8-hour work shift that coincides with an adjacent facility that emits eight hours per day, a worker adjustment factor of 4.2 (24 hours / 8 hours * 7 days / 5 days) is typically used for cancer risk assessment.

A possible problem with using this approach is that wind direction, wind speed, and atmospheric stability can vary throughout the day and night and straight scaling as above may skew the results. As observed in the sensitivity study of the worker adjustment factor (Appendix N), using the 4.2 worker adjustment factor can underestimate the offsite worker's inhalation exposure during nighttime hours. In these cases, it is recommended that the worker adjustment factor be increased to 4.8 (Appendix N) or a more representative offsite worker concentration be processed using the hourly raw results from the air dispersion analysis.

This appendix describes how to calculate refined offsite worker concentrations for a single receptor location using the hourly raw results from the AERMOD air dispersion model. The calculations described in this appendix can be used for assessing acute, 8-hour non-cancer chronic, and inhalation cancer health impacts. The basic steps include 1) determining the averaging periods needed for the offsite worker analysis; 2) outputting the hourly raw results from the AERMOD air dispersion model; 3) extracting the hourly concentrations based on when the receptor is present; and 4) calculating the required averaging periods.

1.0. Determine the Averaging Periods Required for the Offsite Worker Analysis

Before any refined offsite worker concentrations can be calculated, the first step is to determine which type of refined concentrations or refined averaging periods are needed for the analysis. The refined averaging periods needed for the analysis are based on the health value categories that are associated with the pollutants emitted by the facility. Specifically, only pollutants that have inhalation cancer potency factors, 8-hour RELs, and acute RELs can use refined offsite worker concentrations. In addition to the health value category, the type of refined averaging period also depends on if the facility emits continuously or non-continuously. Table M.1 describes the type of refined averaging period needed for assessing inhalation cancer potency factors, 8-hour RELs, and acute RELs. Please note that if calm hour processing is used in the air dispersion analysis, then calm and missing hours must also be considered when calculating the refined concentrations. This process is described further in the subsequent steps of this appendix. Please note that there are restrictions on when an 8-hour REL can be used which are discussed in Chapter 2.

Table M.1. Type of Refined Averaging Period by Health Value Category

Health Value Category	Facility Emission Schedule	Type of Refined Averaging Period
Acute REL	Non-Continuous	The maximum 1-hour concentration that occurs when the facility's emissions and offsite worker's schedules overlap.
	Continuous	
8-Hour REL	Non-Continuous	The sum of the hourly concentrations for every day of the week based on the offsite worker's daily schedule divided by the number of processed hours when the offsite worker is present multiplied X 5/7.
	Continuous	The sum of the hourly concentrations for every day of the week based on the offsite worker's daily schedule divided by the number of processed hours when the offsite worker is present multiplied X 5/7. However, if the offsite worker's shift is less than eight hours (e.g 4 hours) a day, then the number of processed hours should still be based on an 8-hour work shift with air concentration the worker is exposed to during the 4 hours reduced by a factor of 2 and then multiplied X 5/7..
Inhalation Cancer Potency Factor	Non-Continuous	The sum of the hourly concentrations for when the offsite worker is present divided by the total number of hours in a year to give an annual average air concentration
	Continuous	The sum of the hourly concentrations for when the offsite worker is present divided by the number of processed hours for the entire meteorological dataset.

2.0. Output the Hourly Raw Results from AERMOD

The AERMOD input file contains the modeling options, source location and parameter data, receptor locations, meteorological data file specifications, and output options. It is organized into five main sections that include the Control (CO), Source (SO), Receptor (RE), Meteorology (ME), and Output (OU) pathways (U.S. EPA, 2004). In order to output a file suitable for post-processing (i.e., the hourly raw results), the CO, SO, RE, and OU pathways in the AERMOD input file must be modified using a text editor (e.g., Notepad) or third party modeling software.

2.1. *Modify the Control (CO) Pathway to Identify Calm and Missing Hours*

Based on how AERMOD calculates long-term averages (i.e., period average), if calm hour processing is used in the air dispersion analysis, calm and missing hours that occur during the worker's shift must be accounted. Since the hourly raw results from AERMOD do not identify which hours are calm or missing, the Detailed Error Listing File will need to be outputted from the air dispersion analysis. The Detailed Error Listing File will report all calm and missing hours in the air dispersion analysis. The syntax for creating a Detailed Error Listing File in the CO pathway is shown below. The ERRORFIL keyword is followed by a user-defined filename for the output file. An explanation about how this file will be used is in the subsequent sections of this appendix.

Syntax for Creating the Detailed Error Listing File

CO ERRORFIL Filename

2.2. *Modify the Source (SO) Pathway if Unit Emission Rates are used*

In an air dispersion analysis, it is typical to use non-substance specific unit emission rates (e.g., 1 g/s) for evaluating multiple pollutants. This precludes modelers from having to run the air dispersion model for each individual pollutant that is emitted from a source. Unit emission rates allow the air dispersion modeling results to be expressed as dilution factors in $(\mu\text{g}/\text{m}^3)/(\text{g}/\text{s})$. When these dilution factors are combined with the pollutant specific emission rate (g/s), it will yield the actual ground level concentrations $(\mu\text{g}/\text{m}^3)$ for each pollutant in the analysis. However, when there are multiple sources and unit emission rates are used in the air dispersion analysis, the individual source contributions must be provided so the ground level concentrations can be correctly scaled for each pollutant. To do this, the air dispersion input file must be modified to create individual source groups for each source. The example below shows how individual source groups for two sources (S001 and S002) are specified in the SO pathway of an AERMOD input file.

SO STARTING

****S001 and S002 location and source parameters are not shown.****

SRCGROUP SRCGP1 S001
SRCGROUP SRCGP2 S002

This parameter identifies the sources tied to the source group. Use only one source ID per source group.

SO FINISHED

This section specifies the name of your source group. The source group name is what is specified when you output the required concentrations files.

Please note that a separate input file is needed for evaluating acute health impacts when unit emission rates are used and the source has a variable emission schedule (e.g., emissions vary by hour-of-day and day-of-week). Acute health impacts are based on maximum hourly emissions whereas long-term and chronic impacts are based on average hourly emissions. To correctly simulate unit emissions for the acute impacts, a second source with a variable emission rate of “on” (1) or “off” (0) should be used. The example below shows how the variable emission rates should be modified. Alternatively, a source can be duplicated in the same input file instead of rerunning the source using a separate input file.

First Run with Unmodified Emission Rate Factors for Long-Term

EMISFACT	S002	HROFDY	0.000	0.000	0.000	0.000	0.000
	S002	HROFDY	0.000	2.667	2.667	2.667	2.667
	S002	HROFDY	2.667	2.667	1.333	1.333	1.333
	S002	HROFDY	1.333	1.333	1.333	0.000	0.000
	S002	HROFDY	0.000	0.000	0.000	0.000	

Second Run with Modified Emission Rates Factors for Acute

EMISFACT	S002	HROFDY	0.000	0.000	0.000	0.000	0.000
	S002	HROFDY	0.000	1.000	1.000	1.000	1.000
	S002	HROFDY	1.000	1.000	1.000	1.000	1.000
	S002	HROFDY	1.000	1.000	1.000	0.000	0.000
	S002	HROFDY	0.000	0.000	0.000	0.000	

2.3. *Modify the Receptor (RE) Pathway to Reduce the Processing Time*

The POSTFILE option in AERMOD is capable of producing hourly results suitable for post-processing the exposure estimates for the off-site worker. However, without taking appropriate precautions in the parameters of the input file, this option can produce extremely large file sizes especially when evaluating multiple years of meteorological data, a large number of receptors, and short-term averaging periods (e.g., 1-hour). To minimize the amount of processing time and hard disk space, it is recommended to use only a single discrete receptor representing the off-site worker location. Multiple off-site worker locations with the same work shift can also be added as well. The proper syntax for specifying a discrete receptor is shown below. The next section will discuss how to setup the POSTFILE.

Sample Syntax for Creating a Discrete Receptor

`RE DISCCART Xcoord Ycoord (ZelevZhill) (Zflag)`

Please note that depending on how many receptors are specified, a database may be required to sort the hourly results. For example 12 receptors and five years of meteorological data can result in 525,600 records (12 receptors x 5 years x 8760 hours/year).

2.4. *Modify the Output (OU) Pathway to Output the Hourly Raw Results*

To create a file containing the hourly raw results, modify the output pathway to include the POSTFILE keyword and parameters. The sample below shows the syntax for outputting the hourly raw results for a single source. The file generated by this option is suitable for post-processing the acute, inhalation chronic, and inhalation cancer scenarios. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule. Use Table M.2 to help construct the proper syntax for the POSTFILE option. This step must be repeated for each source in the analysis.

Please note that if the data are outputted as binary file, a computer program will be needed to read and parse the data.

Sample Syntax for Outputting the Hourly Concentrations for a Single Source

`OU POSTFILE 1 S001 PLOT PSTS001.TXT`

Table M.2. Descriptions of the POSTFILE Parameters

Keyword	Parameters	
POSTFILE	Aveper Grpid Format Filnam (Funit)	
where:	Aveper	Specifies averaging period to be output to file. Set this value to 1 to output 1-hour raw results.
	Grpid	Specifies source group to be output to file. If there a multiple sources, you will need to repeat the POSTFILE option for each source. You can combine the different outputs to a single file using the Funit parameter.
	Format	Specifies format of file, either UNFORM for binary files or PLOT for formatted files. Unformatted files offer a smaller file size; however, this file requires programming expertise in order to view and parse the data. Selecting the PLOT option will allow you to view the file in any text editor.
	Filnam	Specifies filename for output file
	Funit (optional)	The file unit is an optional parameter. If the filename and the file unit number are the same, the results for different source groups can be combined into a single file.

3.0. Extract the Hourly Concentrations when the Offsite Worker is Present

To calculate the averaging periods for the offsite worker, it is necessary to extract the hourly concentrations based on the offsite worker's schedule. This section provides information on how to extract the hourly concentrations for the offsite worker including the calm and missing hours that may occur during the offsite worker's shift.

At this point, it is recommended the hourly raw results be imported into a spreadsheet or database to assist with the extraction process. The steps that are described below will require additional information for each hourly concentration record. Spreadsheets and database contain preprogrammed functions or allows the use of Structured Query Language to assist with deciphering data.

Please note that if the hourly raw results are imported into a database, familiarity with Structured Query Language and database design is assumed. The steps below will only provide examples for spreadsheets.

3.1. Description of the POSTFILE File Format

AERMOD was compiled using FORTRAN, a type of programming language. When the AERMOD output files are created, it is based on a specified FORTRAN format. The FORTRAN syntax for the POSTFILE format is shown below. The variables provided on each data record in the POSTFILE include the X and Y coordinates of the receptor location, the concentration value for that location, receptor terrain elevation, hill height

scale, flagpole receptor height, the averaging period, the source group ID, and the date for the end of the averaging period (in the form of YYMMDDHH) (U.S. EPA, 2004). Table M.3 shows the equivalent data types based on the POSTFILE format. The equivalent data types shown in Table M.3 can be used as a guide for importing the results into a database or spreadsheet for extracting the offsite worker concentrations. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule (see Figure M.1).

FORTRAN syntax for the POSTFILE Format

(3(1X,F13.5),3(1X,F8.2),2X,A6,2X,A8,2X,I8.8,2X,A8)

Table M.3. POSTFILE Variables and Equivalent Data Types

Column Name	Fortran Format	Equivalent Data Type
X	F13.5	Number/Double Precision
Y	F13.5	Number/Double Precision
AVERAGE_CONC	F13.5	Number/Double Precision
ZELEV	F8.2	Number/Double Precision
ZHILL	F8.2	Number/Double Precision
ZFLAG	F8.2	Number/Double Precision
AVE	A6	6-Character String/Text
GRP	A8	8-Character String/Text
NUM_HRS OR DATE	I8.8	8-Character String/Text
NET_ID	A8	8-Character String/Text

Figure M.1. Sample of an AERMOD POSTFILE

AERMOD (09292): LARGE PS MODELING OPTIONS USED: NonFAULT CONC							08/24/10 07:39:24		
POST/PLOT FILE OF CONCURRENT 1-HR VALUES FOR SOURCE GROUP: S010 FOR A TOTAL OF 1 RECEPTORS. FORMAT: (3(1X,F13.5),3(1X,F8.2),2X,A6,2X,A8,2X,I8.8,2X,A8)									
X	Y	AVERAGE CONC	ZELEV	ZHILL	ZFLAG	AVE	GRP	DATE	NET ID
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010101	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010102	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010103	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010104	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010105	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010106	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010107	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010108	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010109	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010110	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010111	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010112	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010113	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010114	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010115	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010116	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010117	

3.2. Determine the Day-of-Week and Hour-of-Day

In order to extract only the hourly concentrations that occur when offsite worker is present, first determine the day-of-week and hour-of-day for each hourly concentration record. For this step, it is recommended to import the data into a spreadsheet or database. This will allow the addition of columns for the day-of-week and hour-of-day. The purpose of this step is to determine which hourly concentration records are associated with the offsite worker's schedule. This step must be repeated for each source.

To determine the day-of-week and hour-of-day for each record use the date field in the POSTFILE (see Figure M.1). The date field is in the format of YYMMDDHH (e.g., 05010124 equals the data period ending at hour 24 on January 1, 2005). If the data was imported using a spreadsheet, the preprogrammed functions can be used to determine the day-of-week. For example, the WEEKDAY function in Microsoft Excel can be used to determine the day-of-week (See Figure M.3.1). The hour-of-day can simply be extracted using the RIGHT function (See Figure M.3.1).

Please note that in order to use the WEEKDAY function in Microsoft Excel, the date must be first converted into a format that Microsoft Excel can understand. The date field can be converted using the LEFT and MID functions (See Figure M.3).

Figure M.3.1. How to Determine the Day-of-Week and Hour-of-Day in Microsoft Excel

The screenshot shows a Microsoft Excel spreadsheet titled "PSTS010.TXT - Microsoft Excel". The spreadsheet has columns labeled G through M. Column G contains "AVE", H contains "GRP", I contains "DATE", J contains "NET ID", K contains "MDDYY", L contains "DOW", and M contains "HR". Rows 2 through 6 show data for "1-HR" events on "01/01/05".

Three callout boxes provide formulas:

- Formula to convert the date field:**
`=MID("05010101",3,2)&"/"&MID("05010101",5,2)&"/"&LEFT("05010101",2)` will equal 01/01/05
- Formula to determine the day-of-week:**
`=WEEKDAY(01/01/05)` will equal 7 or Saturday (Sun =1, Mon=2, Tues=3, Wed=4, Thurs=5, Fri=6, and Sat=7)
- Formula to determine the hour-of-day:**
`=RIGHT("05010101",2)` will equal 1

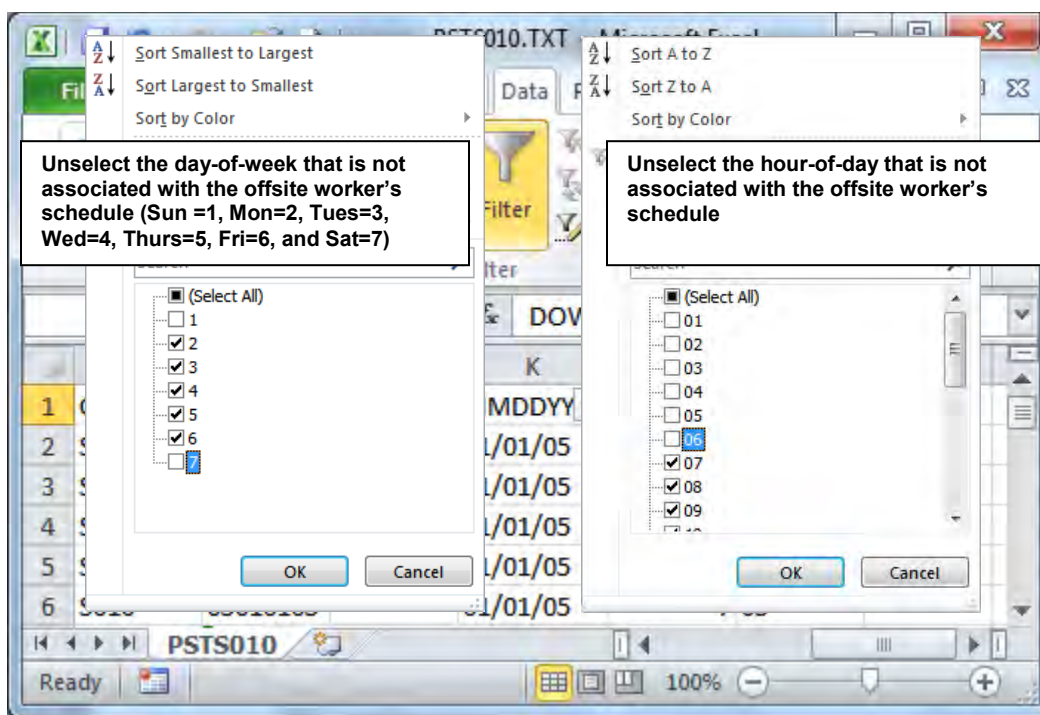
Arrows point from these callouts to the corresponding cells in the spreadsheet: the first callout points to the "DATE" column, the second callout points to the "DOW" column, and the third callout points to the "HR" column.

	G	H	I	J	K	L	M
1	AVE	GRP	DATE	NET ID	MDDYY	DOW	HR
2	1-HR	S010	05010101		01/01/05	7	01
3	1-HR	S010	05010102		01/01/05	7	02
4	1-HR	S010	05010103		01/01/05	7	03
5	1-HR	S010	05010104		01/01/05	7	04
6	1-HR	S010	05010105		01/01/05	7	05

3.3. *Extract the Hourly Concentrations Based on the Offsite Worker's Schedule*

Based on the offsite worker's schedule, filter or query the hourly concentrations using a spreadsheet or database. For example, in Microsoft Excel, you can filter the data by selecting the data filter option (see Figure M.3.2). Then unselect the records that are not associated with the offsite worker's schedule using the day-of-week and hour-of-day fields that were created in previous section. Since the imported data may contain information for multiple receptors, also filter the X and Y coordinates to get the concentrations that are specific to each receptor. The result from the filter will now only show hourly concentrations for when the offsite worker is present. Please note that the day-of-week field may also be filtered for cancer assessment only.

Figure M.3.2. How to Filter the Data in Microsoft Excel



3.4. *Count the Number of Calm and Missing Hours that Occur During the Offsite Worker's Schedule*

If calm hour processing is used in the air dispersion analysis, then calm and missing hours must also be considered when post-processing the long-term average concentrations for the offsite worker. To assist in this calculation, the Detailed Error Listing File that was created from the air dispersion analysis (Section 2.1) can be used to count the number of calm and missing hours that occurred during the worker's shift.

To identify the calm and missing hours, it is recommended to import the Detailed Error Listing File into a spreadsheet or database. Then follow the instructions from Sections 3.2 and 3.3 to determine the number of calm and missing hours that occurs during the offsite worker's schedule. This information is needed to calculate the averaging periods for the offsite worker.

4.0. *Calculate the Refined Averaging Periods for the Offsite Worker Analysis*

Depending on which averaging periods are needed (as determined by Section 1.0), use the sections 4.1 through 4.5 below to calculate refined concentrations for estimating the acute, 8-hour non-cancer chronic, and cancer health impacts. The equations are based on how the maximum 1-hour and period concentrations are calculated in AERMOD. These equations also account for how calm and missing hours are handled by AERMOD (U.S. EPA, 2005). After calculating the appropriate averaging periods, the refined concentrations can be used to assess the health impacts for the offsite worker's inhalation pathway only.

Please note that if unit emission rates were used in the air dispersion analysis, each averaging period calculated using the methods below must be combined with the pollutant specific emission rate (g/s) to yield the actual ground level concentrations ($\mu\text{g}/\text{m}^3$) for each pollutant in the analysis.

4.1. *How to Determine the Maximum 1-Hour Average for a Simple Acute Assessment*

The maximum 1-hour average concentration used to assess the acute health impact represents the highest concentration that occurs when the source's emission schedule and the offsite worker's schedules overlap. To determine the maximum 1-hour average, Sort the extracted hourly concentrations using a spreadsheet or a database. This process must be repeated for each source.

4.2. Equation for Calculating the Average Concentration for the 8-Hour Non-Cancer Chronic from a Non-Continuous Emitting Source

Below is the equation for calculating the average concentration for the 8-hour chronic assessments. This calculation must be repeated for each source.

$$C_{worker_period_average} = \frac{\sum C_{hourly}}{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}}$$

Where:

C_{hourly} = the concentration that occurs during the worker's daily shift. This also includes every day of the week regardless if the worker is present.

N_{total_hrs} = the number of processed hours that occurs during the worker's shift. This includes every day of the week regardless if the worker is present.

N_{calm_hrs} = the number of calm hours that occurs that occurs during the worker's shift. This includes every day of the week regardless if the worker is present

$N_{missing_hrs}$ = the number of missing hours that occurs during the worker's shift. This includes every day of the week regardless if the worker is present

4.3. Equation for Calculating the Average Concentration for the 8-Hour Non-Cancer Chronic from a Continuous Emitting Source

Below is the equation for calculating the average concentration for the 8-hour chronic assessments. This calculation must be repeated for each source.

$$C_{worker_period_average} = \frac{\sum C_{hourly}}{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}}$$

Where:

C_{hourly} = the concentration that occurs during the worker's daily shift. This includes every day of the week regardless if the worker is present.

N_{total_hrs} = the number of processed hours that occurs during the worker's shift. This includes every day of the week regardless if the worker is present. However, if the offsite worker's shift is less than eight hours a day, then the number of processed hours should still be based on an 8-hour work shift. For example, if a worker's shift only last six hours a day, the number of processed hours should be eight (8) multiplied by the number of days in the meteorological dataset.

N_{calm_hrs} = the number of calm hours that occurs that occurs during the worker's shift. This includes every day of the week regardless if the worker is present

$N_{missing_hrs}$ = the number of missing hours that occurs during the worker's shift. This includes every day of the week regardless if the worker is present

4.4. Equation for Calculating the Period Average for the Inhalation Cancer Pathway from a Non-Continuous Emitting Source

Below is the equation for calculating the period average for the inhalation cancer pathway. This calculation must be repeated for each source.

$$C_{worker_period_average} = \frac{\sum C_{hourly}}{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}}$$

Where:

C_{hourly} = the concentration that occur during the worker's shift

N_{total_hrs} = the number of processed hours that occurs during worker's shift

N_{calm_hrs} = the number of calm hours that occurs during the worker's shift

$N_{missing_hrs}$ = the number of missing hours that occurs during worker's shift

4.5. Equation for Calculating the Period Average for the Inhalation Cancer Pathway from a Continuous Emitting Source

Below is the equation for calculating the period average for the inhalation cancer pathway. This calculation must be repeated for each source.

$$C_{worker_period_average} = \frac{\sum C_{hourly}}{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}}$$

Where:

C_{hourly} = the concentration that occur during the worker's shift

N_{total_hrs} = the number of processed hours for the entire meteorological dataset

N_{calm_hrs} = the number of calm hours for the entire meteorological dataset

$N_{missing_hrs}$ = the number of missing hours for the entire meteorological dataset

REFERENCES

U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model – AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Guideline on Air Quality Models (Revised). 40 CFR 51, Appendix W.

Appendix N.

Sensitivity Study of the Worker Adjustment Factor using AERMOD

N.1. Introduction

The offsite worker health risk analysis begins with estimating the pollutant concentration at a receptor location. To estimate this concentration, the typical approach is to use the residential annual concentration that is modeled based on the adjacent facility's emission schedule. However, if the facility emissions are non-continuous, the residential concentration may not represent what the worker breathes during their work shift. In lieu of conducting additional special case modeling which can be time-consuming, the residential annual concentration is adjusted upwards using a worker adjustment factor based on the facility's emission schedule with respect to the worker's schedule. For an 8-hour work shift that coincides with an adjacent facility that emits eight hours per day, a worker adjustment factor of 4.2 ($24 \text{ hours} / 8 \text{ hours} * 7 \text{ days} / 5 \text{ days}$) is typically used for cancer risk assessment.

A possible problem with using this approach is that wind direction, wind speed, and atmospheric stability can vary throughout the day and night and straight scaling as above may skew the results. If the diurnal variation is considerable, the 4.2 adjustment could be an under- or overestimate depending on the time of day that the offsite worker shift begins and ends. The goal of this study is to test the validity of the 4.2 adjustment using five meteorological data sets from five different locations in California and with three different size point sources. The modeling is performed done with 8-hour emissions coinciding with the offsite workers schedule. The 8-hour shifts are modeled as starting every hour around the clock.

To perform this study, the AERMOD air dispersion model, meteorological data from five locations (i.e., Kearny Mesa, Palomar, Pomona, Redlands, and San Bernardino), and three different size point sources (small, medium, and large) are used. The AERMOD-ready meteorological datasets are selected to represent a range of meteorological conditions around the state. To mirror the assumptions used in the 4.2 worker adjustment factor, the emission rate of each source are simulated for eight continuous hours with 24 different start times for five days a week (Monday through Friday). This will simulate the conditions that result during an 8-hour work schedule starting any hour of the day. In addition, the emitting source and offsite worker are assumed to have coincident schedules.

Using the AERMOD air dispersion modeling results, the Point of Maximum Impact (PMI) is identified and the hourly raw concentrations are post-processed to calculate the long-term offsite worker concentration for each scenario. To test the validity of the worker adjustment factor, the calculated long-term offsite worker concentration is divided by the long term residential average to obtain a quotient that is unique to each meteorological data location. The quotient is then compared to the 4.2 worker adjustment factor to see which is higher or more health protective.

Although this study is primarily based on an 8-hour work schedule, the actual duration that an offsite worker is present near the emitting source may vary when considering a lunch break or a longer work shift. Thus, 10-hour scenarios are also evaluated. The worker adjustment factor for ten hours is 3.4 ($24 \text{ hours} / 10 \text{ hours} * 7 \text{ days} / 5 \text{ days}$).

N.2. Background on the Worker Adjustment Factor for Inhalation Cancer Assessments

There are basically two approaches that can be used to calculate the offsite worker inhalation exposure for cancer assessments. One approach is to post-process the hourly dispersion modeling results and examine the coincident hours between the source's emission schedule and the worker's schedule. The second, and more commonly used approach, is to apply a worker adjustment factor to the modeled long-term residential concentration. While post-processing the hourly modeling output will offer a more representative worker concentration, it is very time consuming and requires the management of large amounts of data. Thus, the simplistic approach of applying a worker adjustment factor to estimate the worker inhalation exposure is typically used.

The worker adjustment factor is used together with the long-term residential concentration to estimate the offsite worker's inhalation exposure. This calculation is summarized below.

- a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year or multi-year analysis are used).
- b. Determine the coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule.
- c. Calculate the worker adjustment factor using Equation N.1. When assessing inhalation cancer health impacts, a discount factor (*DF*) may also be applied if the offsite worker's schedule partially overlaps with the source's emission schedule. The discount factor is based on the number of coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule (see Equation N.2).

Please note that worker adjustment factor does not apply if the source's emission schedule and the offsite worker's schedule do not overlap. Since the worker is not around during the time that the source is emitting, the worker is not exposure to the source's emission (i.e., the DF in Equation N.2 becomes 0).

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} \times DF$$

Eq. N.1

Where:

WAF = the worker adjustment factor

$H_{residential}$ = the number of hours per day the long-term residential concentration is based on (24)

H_{source} = the number of hours the source operates per day

$D_{residential}$ = the number of days per week the long-term residential concentration is based on (7).

D_{source} = the number of days the source operates per week.

DF = a discount factor for when the offsite worker's schedule partially overlaps the source's emission schedule.

Use 1 if the offsite worker's schedule occurs within the source's emission schedule. If the offsite worker's schedule partially overlaps with the source's emission schedule, then calculate the discount factor using Equation N.2 below.

$$DF = \frac{H_{coincident}}{H_{worker}} \times \frac{D_{coincident}}{D_{worker}}$$

Eq. N.2

Where:

DF = the discount factor for assessing cancer impacts

$H_{coincident}$ = the number of hours per day the offsite worker's schedule and the source's emission schedule overlap

$D_{coincident}$ = the number of days per week the offsite worker's schedule and the source's emission schedule overlap.

H_{worker} = the number of hours the offsite worker works per day

D_{worker} = the number of days the offsite worker works per week.

- d. The final step is to estimate the offsite worker inhalation exposure by multiplying the worker adjustment factor with the long-term residential concentration.

N.3. Method and Modeling Parameters

For this study, all scenarios are simulated using the AERMOD (Version 09292) air dispersion model. The modeling parameters input to AERMOD and methods used to process the model outputs are discussed below.

N.3.1. Point Source Release Parameters

This study uses three different size point sources representing small, medium, and large. The point source release parameters are shown in Table N.1.

Table N.1. Point Source Modeling Parameters

Source Size	Emission Rate (g/s)	Release Ht (m)	Diameter (m)	Exit Temp (K)	Exit Vel (m/s)	Building Dimensions L (m) x W (m) x H (m)	XBADJ YBADJ ¹
Large	1	30	3	400	10	15 x 15 x 6	7.5
Medium	1	10	1	400	10	12 x 12 x 6	6
Small	1	2.15	0.1	400	10	6 x 6 x 2	3

1 – The XBADJ and YBADJ are keywords defining the along-flow and across-flow distances from the stack to the center of the upwind face of the projected building, respectively (U.S. EPA, 2004).

N.3.2. Temporal Emission Rate

Each point source (i.e., small, medium, and large) is simulated with continuous emissions for eight hours a day from Monday through Friday. In addition, all starting hour combinations (24 scenarios) are evaluated by duplicating each source 24 times with unique start times. Table N.2 shows the 8-hour operating schedule for each scenario. All emissions for Saturday and Sunday are set at zero. This process will also be repeated for the 10-hour evaluation. Table N.3 shows the 10-hour operating schedule for each scenario.

Table N.2. 8-Hour Operating Schedule

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON																	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON																	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON																	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON																	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON																	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON																	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON																	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON																ON
8:00 AM		ON	ON	ON	ON	ON	ON	ON	ON															
9:00 AM			ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM				ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM					ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM						ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM							ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM								ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM									ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM										ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM											ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM												ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM													ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM														ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM															ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM																ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM																	ON	ON	ON	ON	ON	ON	ON	ON

Table N.3. 10-Hour Operating Schedule

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON															ON	ON	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON															ON	ON	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON															ON	ON	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON															ON	ON	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON															ON	ON	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON															ON	ON	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON															ON	ON	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON															ON	ON
8:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON															ON
9:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM		ON	ON	ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM			ON	ON	ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM				ON	ON	ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM					ON	ON	ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM						ON	ON	ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM							ON	ON	ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM								ON	ON	ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM									ON	ON	ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM										ON	ON	ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM											ON	ON	ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM												ON	ON	ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM													ON	ON	ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM														ON	ON	ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM															ON	ON	ON	ON	ON	ON	ON	ON	ON	ON

N.3.3. Receptor Grid Parameters

A 1000 meter by 1000 meter receptor grid is centered over each source. The receptors are spaced in 50 meter increments resulting in 441 receptor points. All receptor flagpole heights are set at 1.2 meters above ground.

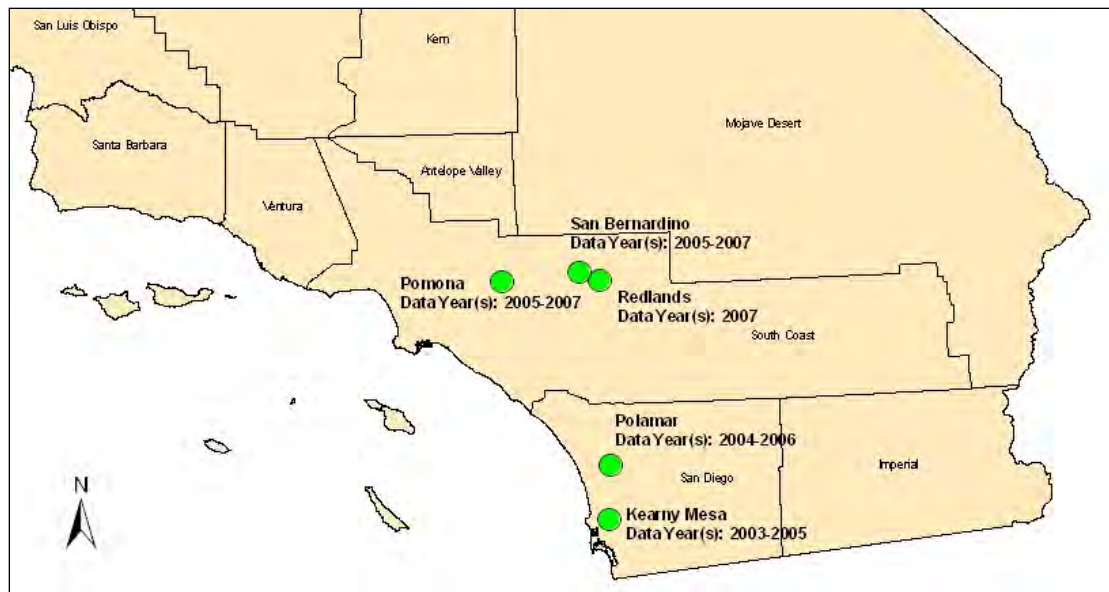
N.3.4. Meteorological Data

The meteorological data input to AERMOD were requested from two local air districts in California (ARB 2009a and ARB 2009b). The meteorological data that were provided by the Districts are, based on the Districts' observations and expertise, datasets that were likely to result in higher than average long-term impacts. The data includes four multi-year files and one single year file. Table N.4 shows the meteorological datasets used in this study. Figure N.1 shows the location of the meteorological station. The AERMOD profile base is defaulted to 10 meters above mean sea level for each meteorological file.

Table N.4. Meteorological Datasets

Data Provider	Area	Data Year(s)	Total Hours	Percent of Calm and Missing Hours	Avg. Wind Speed (m/s)
San Diego Air Pollution Control District	Kearny Mesa	2003-2005	26304	6.9	1.36
	Palomar	2004-2006	26304	8.7	1.36
South Coast Air Quality Management District	Pomona	2005-2007	26280	1.6	1.18
	Redlands	2007	8760	5.5	0.94
	San Bernardino	2005-2007	26280	4.9	1.44

Figure N.1. Meteorological Data Set Locations



N.3.5. Post-Processing the Period Average Concentrations for the Offsite Worker

The period average concentration represents the average concentration of all hours processed within the meteorological set. Equation N.3 shows how the period average is calculated in AERMOD including how calm and missing hours are processed (U.S. EPA, 2005).

$$C_{period_average} = \frac{\sum C_{hourly}}{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}} \quad \text{Eq. N.3}$$

Where:

C_{hourly} = the concentration that occurs at a given hour
 N_{total_hrs} = the number of processed hours reported by AERMOD (e.g., 1 yr = 8760 hours)
 N_{calm_hrs} = the number of calm hours reported by AERMOD
 $N_{missing_hrs}$ = the number of missing hours reported by AERMOD

Normally to post-process hourly data, the off-site worker hours are extracted from the hourly model output files and then averaged. However, this sensitivity study assumes the hourly emissions are coincident with the off-site worker schedule. Since this is the case, the 8-hour period average for the offsite worker can simply be scaled from the period average reported by AERMOD (see Equation N.4). To make sure this calculation is accurate; a check was performed by processing the hourly concentrations for one receptor with the Pomona data. If the emission schedule was not 100% coincident with the offsite worker, then all post-processing would have to be completed on an hourly basis. See Appendix M for more information on how to post-process worker concentrations using hourly raw results.

$$C_{worker_period_average} = C_{period_average} \times \frac{N_{total_hrs} - N_{calm_hrs} - N_{missing_hrs}}{N_{worker_hrs} - N_{worker_calm_hrs} - N_{worker_missing_hrs}} \quad \text{Eq. N.4}$$

Where:

$C_{period_average}$ = the period concentration reported by AERMOD
 N_{total_hrs} = the total number of processed hours reported by AERMOD
 N_{calm_hrs} = the total number of calm hours reported by AERMOD
 $N_{missing_hrs}$ = the total number of missing hours reported by AERMOD
 $N_{worker_hrs}^a$ = the total number of hours that occurred during the worker's shift
 $N_{worker_calm_hrs}^b$ = the number of calm hours that occurs during the worker's shift
 $N_{worker_missing_hrs}^b$ = the number of missing hours that occurred during the worker's shift

a – The worker hours are determined by multiplying the number of weekdays (Monday through Friday) that occurs in the meteorological data set by the work shift duration (8 hours). For example, a meteorological data set ranging from 1/1/2003 to 12/31/2005 contains 783 weekdays. If you multiply the number weekdays by the work shift duration (8 hour/day), this will equal 6264 worker hours. The number of weekdays varies depending on the day of the week January 1st starts on.

b – Calm and missing hours are reported in the AERMOD Detailed Message Listing File. To determine the number of worker calm and missing hours, the calm and missing hours that occur during the worker shift are isolated and summed.

N.4. Results

To test the validity of the worker adjustment factor, the post-processed period average concentration for the offsite worker was divided by the modeled period residential average to obtain a quotient. This calculation was performed at the PMI of each scenario. If the quotient is smaller or equal to the worker adjustment factor, the worker adjustment factor is considered a suitable health protective approximation. If the quotient is greater, the worker adjustment factor will underestimate the long-term average concentration and would not be the most conservative estimation of what the worker breathes. For these scenarios, the 8-hour and 10-hour worker adjustment factors are 4.2 and 3.4, respectively. The results for this study are summarized in the figures and tables below. To view the details for every scenario, see Appendix N-1.

Figure N.2 shows how the post-processed period averages changes over 8-hour rolling work shifts. The value at each 8-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 4.2 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five meteorological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 8 am and 3 pm (i.e., 8-hour work shifts starting at 8 am and ending by 11 pm).

Figure N.2. Summary of the 8-Hour Scenarios

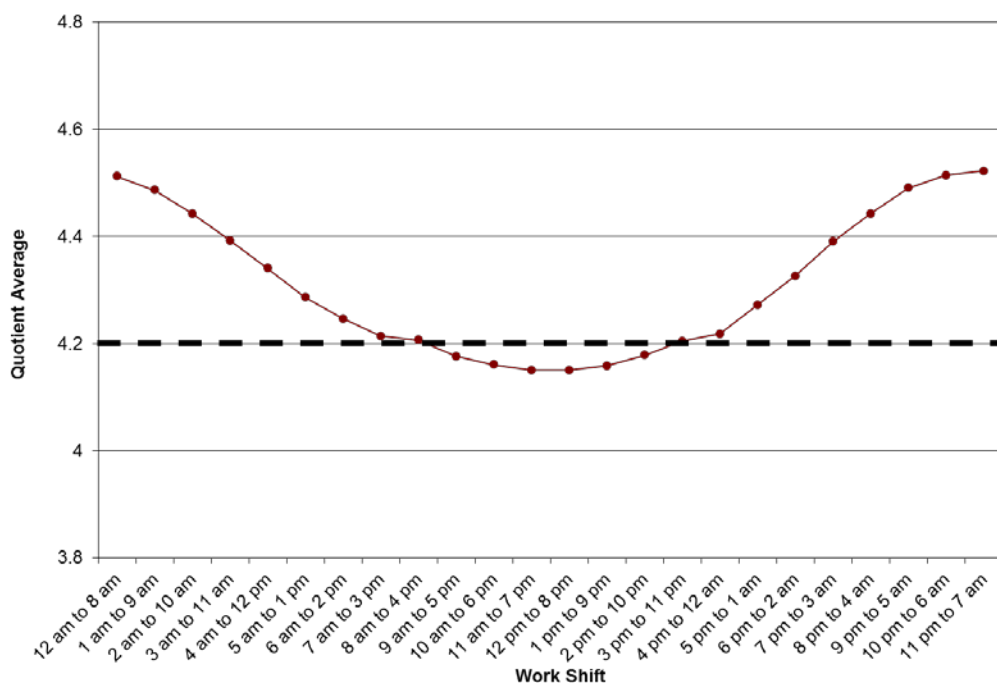


Figure N.3 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 8-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.3. Average Percent of Calm and Missing Hours for 8-Hour Work Shifts

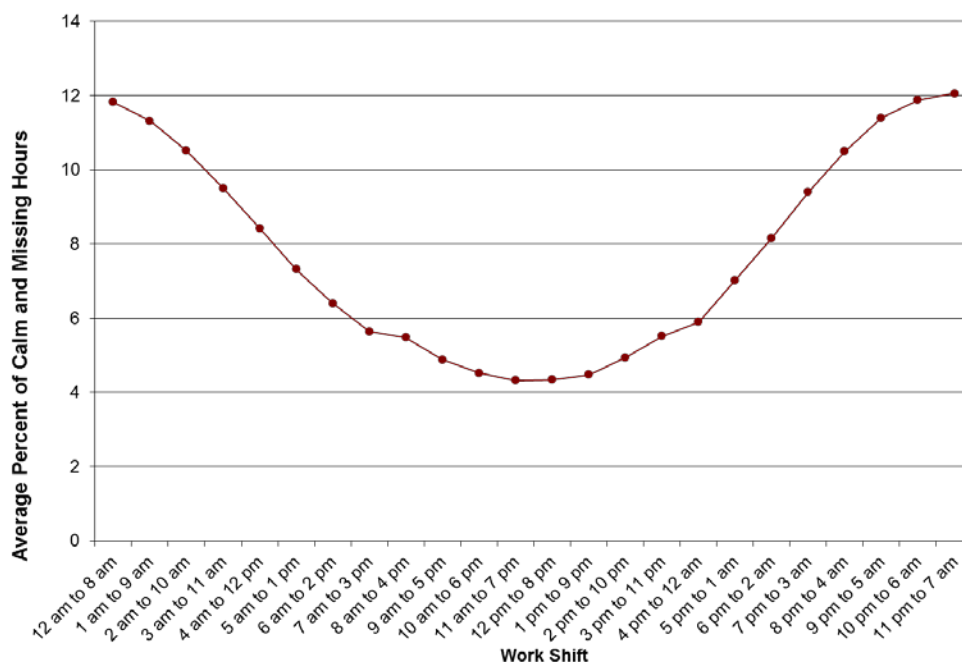


Figure N.4 shows how the post-processed period averages changes over 10-hour rolling work shifts. The value at each 10-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 3.4 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five meteorological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 5 am and 4 pm (i.e., 10-hour work shifts starting at 5 am and ending by 2 am).

Figure N.4. Summary of the 10-Hour Scenarios

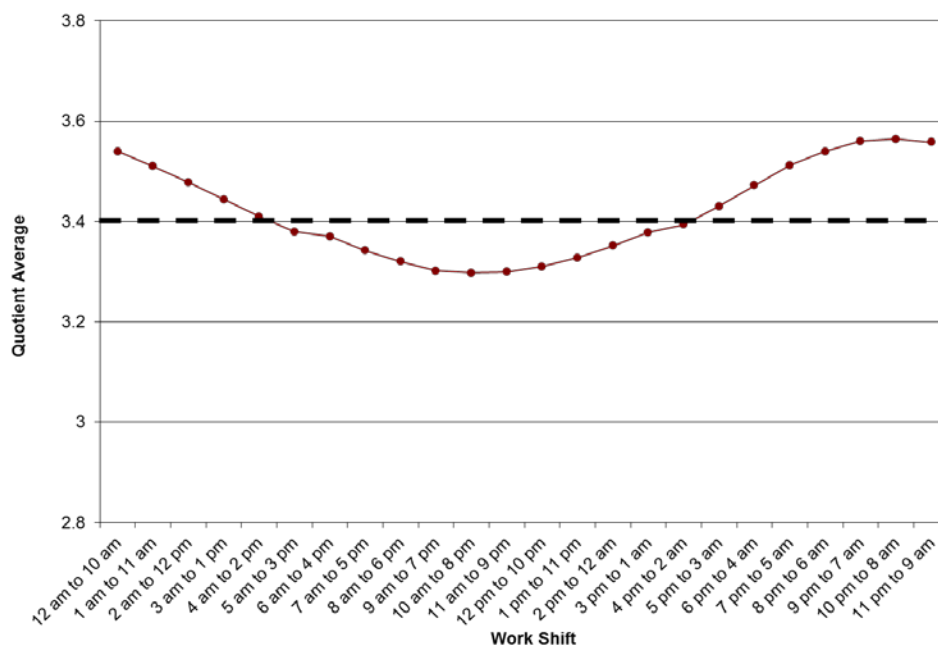


Figure N.5 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 10-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.5. Average Percent of Calm and Missing Hours for 10-Hour Work Shifts

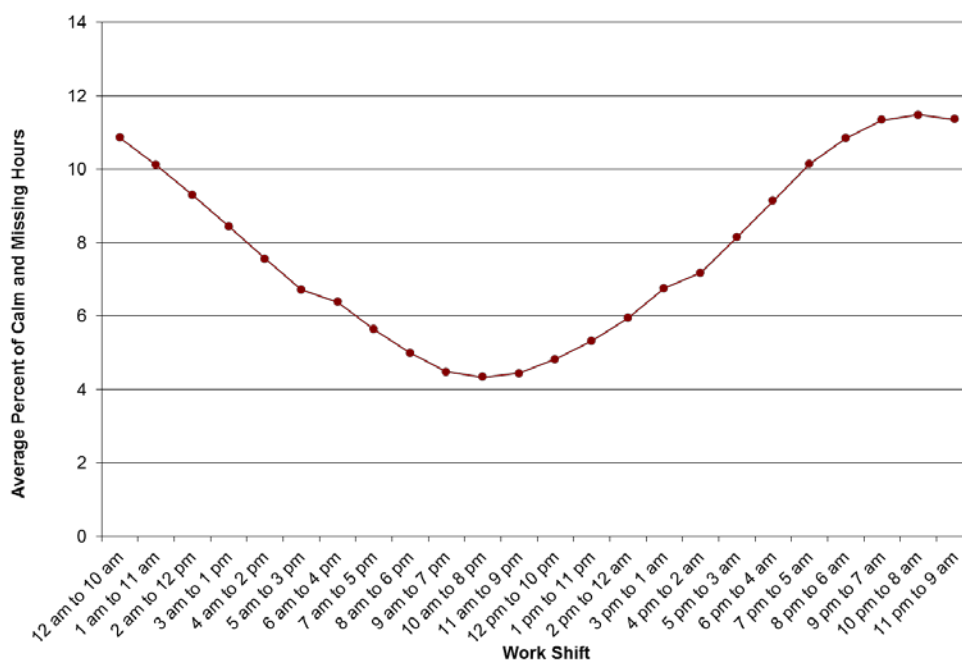


Table N.5 shows the average, minimum, and maximum quotients across all 24 8-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

Table N.5. Summary of the Average 8-Hour Scenarios by Point Source Size

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	9.6 (6.8 to 11.8)
Palomar	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	12.2 (8.2 to 17.5)
Pomona	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	2.3 (2.1 to 2.5)
Redlands	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	7.6 (1.0 to 16.5)
San Bernardino	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	6.9 (1.4 to 14.1)

Table N.6 shows the average, minimum, and maximum quotients across all 24 10-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

Table N.6. Summary of the Average 10-Hour Scenarios by Point Source Size

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	9.6 (7.5 to 11.6)
Palomar	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	12.2 (8.0 to 17.1)
Pomona	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	2.3 (2.2 to 2.5)
Redlands	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	7.6 (1.1 to 15.2)
San Bernardino	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	6.9 (1.5 to 13.1)

N.5. Conclusions

The goal of this study was to determine if the worker adjustment factor of 4.2 (8hours/day, 5 days/week) or 3.4 (10 hours/day, 5 days/week) would always yield a more conservative or health protective approximation using five meteorological data sets. This study demonstrated that the worker adjustment factor does not always represent the most health protective approximation of long-term hourly model predictions. This is primarily observed during night conditions. Air Districts may wish to evaluate their meteorological data to determine an appropriate worker adjustment factor for their area using the methods described in this appendix.

Although the meteorological data used in this study are site-specific, several general conclusions and recommendations can be made. These conclusions and recommendations are summarized below.

- ***The worker adjustment factor is generally a suitable health protective approximation for daytime work shifts.***

For the meteorological data used in this study, the results show that the worker adjustment factor is a suitable health protective approximation for work shifts that occur during the daytime hours. When comparing the 8-hour and 10-hour scenarios, the results show the range of work shifts that were considered a more health protective approximation increased with the longer work shift duration.

- ***The size of the emitting source did not affect the long-term concentration approximated with the worker adjustment factor.***

The size of the source was inconsequential in determining whether the worker adjustment factor is health protective. This is because the worker adjustment factor is applied to the modeling results after the air dispersion analysis has been completed. However, it should be noted that the size of the source does affect the location of the PMI during a specific time of day. This is shown in the scenario details in Appendix N-1.

- ***The worker adjustment factor may not represent the most conservative estimation of the worker's inhalation exposure for nighttime work shifts.***

In most cases, the worker adjustment factor will represent a health protective approximation for work shifts that occur during the daytime. However, the worker adjustment factor may not represent the most conservative estimation when the source's emission schedule and offsite worker's schedules are 100% coincident at night. It is recommended that the offsite worker long-term average concentrations be post-processed using the hourly dispersion modeling results when examining work shifts occurring at night. Alternatively, a more conservative worker adjustment factor can be used to account for the calm hours (see the next bullet point below).

- ***Recommended worker adjustment factor for 8 and 10-hour work shifts***

Based on the five meteorological data sets used in this study, the range of worker adjustment factors (WAF) was observed between 4.2 and 4.8. We recommend using the 4.2 WAF for most cases. In the event of predominant night time emissions and worker schedule or if only one year of meteorological data are available, then we recommend using 4.8 for the 8-hour WAF.

N.6. REFERENCES

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APPENDIX N-1 – SCENARIO DATA DETAILS

KEARNY MESA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	500	0.02584	26304	1813	632.84744	6264	723	11.5	0.11421	4.42
2	0	300	0.05638	26304	1813	1380.80258	6264	739	11.8	0.24992	4.43
3	150	-150	0.10366	26304	1813	2538.73706	6264	729	11.6	0.45867	4.42
4	150	-100	0.19993	26304	1813	4896.48563	6264	718	11.5	0.88289	4.42
5	200	-100	0.33363	26304	1813	8170.93233	6264	700	11.2	1.46854	4.40
6	200	-100	0.48136	26304	1813	11788.98776	6264	688	11.0	2.11424	4.39
7	200	-100	0.62685	26304	1813	15352.18335	6264	684	10.9	2.75129	4.39
8	200	-100	0.76245	26304	1813	18673.16295	6264	681	10.9	3.34465	4.39
9	200	-100	0.85443	26304	1813	20925.84513	6264	665	10.6	3.73743	4.37
10	250	-100	0.89012	26304	1813	21799.92892	6264	618	9.9	3.86113	4.34
11	250	-100	0.85448	26304	1813	20927.06968	6264	568	9.1	3.67399	4.30
12	250	-100	0.76187	26304	1813	18658.95817	6264	517	8.3	3.24673	4.26
13	250	-100	0.63409	26304	1813	15529.49819	6264	488	7.8	2.68863	4.24
14	250	-100	0.48738	26304	1813	11936.42358	6264	467	7.5	2.05907	4.22
15	300	-150	0.34902	26304	1813	8547.84882	6264	454	7.2	1.47123	4.22
16	300	-150	0.20978	26304	1813	5137.72198	6264	433	6.9	0.88110	4.20
17	300	-150	0.09739	26304	1813	2385.17849	6264	425	6.8	0.40849	4.19
18	350	-200	0.02843	26304	1813	696.27913	6264	456	7.3	0.11988	4.22
19	0	500	0.00479	26304	1813	117.31189	6264	516	8.2	0.02041	4.26
20	-50	500	0.00491	26304	1813	120.25081	6264	578	9.2	0.02115	4.31
21	0	500	0.00512	26304	1813	125.39392	6264	625	10.0	0.02224	4.34
22	0	500	0.00513	26304	1813	125.63883	6264	658	10.5	0.02241	4.37
23	0	500	0.00528	26304	1813	129.31248	6264	675	10.8	0.02314	4.38
24	0	500	0.01002	26304	1813	245.39982	6264	699	11.2	0.04410	4.40

KEARNY MESA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	100	0.48213	26304	1813	11807.84583	6264	723	11.5	2.13100	4.42
2	0	100	0.99949	26304	1813	24478.50959	6264	739	11.8	4.43050	4.43
3	50	50	1.69544	26304	1813	41523.02104	6264	729	11.6	7.50190	4.42
4	50	50	2.6458	26304	1813	64798.28780	6264	718	11.5	11.68379	4.42
5	50	50	3.51528	26304	1813	86092.72248	6264	700	11.2	15.47317	4.40
6	50	50	4.24949	26304	1813	104074.25959	6264	688	11.0	18.66468	4.39
7	100	-50	5.33685	26304	1813	130704.79335	6264	684	10.9	23.42380	4.39
8	100	-50	6.51541	26304	1813	159568.90631	6264	681	10.9	28.58121	4.39
9	100	-50	7.325	26304	1813	179396.57500	6264	665	10.6	32.04082	4.37
10	100	-50	7.60514	26304	1813	186257.48374	6264	618	9.9	32.98928	4.34
11	100	-50	7.28086	26304	1813	178315.54226	6264	568	9.1	31.30540	4.30
12	100	-50	6.51093	26304	1813	159459.18663	6264	517	8.3	27.74651	4.26
13	100	-50	5.53256	26304	1813	135497.92696	6264	488	7.8	23.45878	4.24
14	100	-50	4.37499	26304	1813	107147.88009	6264	467	7.5	18.48333	4.22
15	100	-50	3.13098	26304	1813	76680.83118	6264	454	7.2	13.19808	4.22
16	100	-50	1.92339	26304	1813	47105.74449	6264	433	6.9	8.07850	4.20
17	150	-50	0.97341	26304	1813	23839.78431	6264	425	6.8	4.08285	4.19
18	200	-100	0.37344	26304	1813	9145.91904	6264	456	7.3	1.57471	4.22
19	0	150	0.19509	26304	1813	4777.94919	6264	516	8.2	0.83124	4.26
20	0	150	0.18348	26304	1813	4493.60868	6264	578	9.2	0.79029	4.31
21	0	150	0.17623	26304	1813	4316.04893	6264	625	10.0	0.76539	4.34
22	0	150	0.16448	26304	1813	4028.27968	6264	658	10.5	0.71857	4.37
23	0	150	0.16295	26304	1813	3990.80845	6264	675	10.8	0.71405	4.38
24	0	150	0.22443	26304	1813	5496.51513	6264	699	11.2	0.98769	4.40

KEARNY MESA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	56.94704	26304	1813	1394689.95664	6264	723	11.5	251.70366	4.42
2	0	50	63.90855	26304	1813	1565184.29805	6264	739	11.8	283.29128	4.43
3	0	50	72.78622	26304	1813	1782607.31402	6264	729	11.6	322.06094	4.42
4	0	50	80.59339	26304	1813	1973812.71449	6264	718	11.5	355.89843	4.42
5	0	50	86.44869	26304	1813	2117214.86679	6264	700	11.2	380.52029	4.40
6	50	0	96.25147	26304	1813	2357294.75177	6264	688	11.0	422.75731	4.39
7	50	0	117.66867	26304	1813	2881823.39697	6264	684	10.9	516.45581	4.39
8	50	0	138.64904	26304	1813	3395653.63864	6264	681	10.9	608.21308	4.39
9	50	0	156.76654	26304	1813	3839369.33114	6264	665	10.6	685.72412	4.37
10	50	0	172.75048	26304	1813	4230832.00568	6264	618	9.9	749.35034	4.34
11	50	0	184.10847	26304	1813	4509000.53877	6264	568	9.1	791.60824	4.30
12	50	0	190.80885	26304	1813	4673099.54535	6264	517	8.3	813.13721	4.26
13	50	0	183.97723	26304	1813	4505786.33993	6264	488	7.8	780.08766	4.24
14	50	0	168.91026	26304	1813	4136781.17766	6264	467	7.5	713.60724	4.22
15	50	0	150.42213	26304	1813	3683988.38583	6264	454	7.2	634.07717	4.22
16	50	-50	146.48297	26304	1813	3587514.41827	6264	433	6.9	615.24857	4.20
17	50	-50	144.08415	26304	1813	3528764.91765	6264	425	6.8	604.34405	4.19
18	50	-50	130.6006	26304	1813	3198539.29460	6264	456	7.3	550.71269	4.22
19	50	-50	111.9118	26304	1813	2740831.89380	6264	516	8.2	476.83227	4.26
20	50	-50	86.25428	26304	1813	2112453.57148	6264	578	9.2	371.51839	4.31
21	50	-50	65.37008	26304	1813	1600978.62928	6264	625	10.0	283.91180	4.34
22	0	50	56.60048	26304	1813	1386202.35568	6264	658	10.5	247.27120	4.37
23	0	50	53.20196	26304	1813	1302969.20236	6264	675	10.8	233.13101	4.38
24	-100	-100	54.24037	26304	1813	1328400.90167	6264	699	11.2	238.70636	4.40

PALOMAR - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	250	0.02363	26304	2291	567.42719	6256	1096	17.5	0.10997	4.65
2	100	150	0.0631	26304	2291	1515.22030	6256	1090	17.4	0.29331	4.65
3	150	50	0.14317	26304	2291	3437.94121	6256	1050	16.8	0.66038	4.61
4	150	50	0.27432	26304	2291	6587.24616	6256	971	15.5	1.24640	4.54
5	200	50	0.42859	26304	2291	10291.73167	6256	879	14.1	1.91403	4.47
6	200	50	0.58751	26304	2291	14107.87763	6256	788	12.6	2.58008	4.39
7	200	0	0.73867	26304	2291	17737.68271	6256	701	11.2	3.19310	4.32
8	200	0	0.87304	26304	2291	20964.30952	6256	628	10.0	3.72500	4.27
9	250	0	0.96493	26304	2291	23170.86409	6256	679	10.9	4.15472	4.31
10	250	0	0.99791	26304	2291	23962.81283	6256	589	9.4	4.22848	4.24
11	250	0	0.9484	26304	2291	22773.92920	6256	540	8.6	3.98424	4.20
12	250	0	0.83614	26304	2291	20078.22982	6256	518	8.3	3.49917	4.18
13	250	0	0.68595	26304	2291	16471.71735	6256	517	8.3	2.87014	4.18
14	250	0	0.51501	26304	2291	12366.93513	6256	523	8.4	2.15715	4.19
15	300	0	0.34888	26304	2291	8377.65544	6256	550	8.8	1.46822	4.21
16	300	-50	0.20229	26304	2291	4857.58977	6256	596	9.5	0.85823	4.24
17	300	-100	0.10109	26304	2291	2427.47417	6256	516	8.2	0.42290	4.18
18	300	-150	0.0311	26304	2291	746.80430	6256	612	9.8	0.13232	4.25
19	-450	-200	0.00583	26304	2291	139.99579	6256	701	11.2	0.02520	4.32
20	-400	-150	0.00576	26304	2291	138.31488	6256	802	12.8	0.02536	4.40
21	-400	-200	0.00503	26304	2291	120.78539	6256	895	14.3	0.02253	4.48
22	-400	-200	0.00427	26304	2291	102.53551	6256	980	15.7	0.01943	4.55
23	-400	-200	0.00323	26304	2291	77.56199	6256	1040	16.6	0.01487	4.60
24	-500	-500	0.0081	26304	2291	194.50530	6256	1067	17.1	0.03748	4.63

PALOMAR - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	50	0.39916	26304	2291	9585.02908	6256	1096	17.5	1.85756	4.65
2	50	50	1.1355	26304	2291	27266.76150	6256	1090	17.4	5.27812	4.65
3	50	50	2.23922	26304	2291	53770.38986	6256	1050	16.8	10.32854	4.61
4	50	50	3.46481	26304	2291	83200.48253	6256	971	15.5	15.74276	4.54
5	100	0	5.01511	26304	2291	120427.83643	6256	879	14.1	22.39685	4.47
6	100	0	7.1387	26304	2291	171421.60310	6256	788	12.6	31.34996	4.39
7	100	0	9.3361	26304	2291	224187.76930	6256	701	11.2	40.35783	4.32
8	100	0	11.30065	26304	2291	271362.50845	6256	628	10.0	48.21651	4.27
9	100	0	12.55274	26304	2291	301428.94562	6256	679	10.9	54.04858	4.31
10	100	0	12.9907	26304	2291	311945.67910	6256	589	9.4	55.04600	4.24
11	100	0	12.32253	26304	2291	295900.91289	6256	540	8.6	51.76713	4.20
12	100	0	10.99232	26304	2291	263958.58016	6256	518	8.3	46.00184	4.18
13	100	0	9.16435	26304	2291	220063.53655	6256	517	8.3	38.34528	4.18
14	100	0	7.04288	26304	2291	169120.67744	6256	523	8.4	29.49951	4.19
15	100	0	4.85232	26304	2291	116518.76016	6256	550	8.8	20.42039	4.21
16	100	0	2.83666	26304	2291	68116.71658	6256	596	9.5	12.03476	4.24
17	150	0	1.4789	26304	2291	35512.82570	6256	516	8.2	6.18690	4.18
18	150	0	0.51952	26304	2291	12475.23376	6256	612	9.8	2.21035	4.25
19	500	100	0.16252	26304	2291	3902.59276	6256	701	11.2	0.70254	4.32
20	-100	-50	0.13578	26304	2291	3260.48514	6256	802	12.8	0.59782	4.40
21	-100	-50	0.12284	26304	2291	2949.75692	6256	895	14.3	0.55023	4.48
22	-100	-50	0.10491	26304	2291	2519.20383	6256	980	15.7	0.47748	4.55
23	-150	-50	0.08895	26304	2291	2135.95635	6256	1040	16.6	0.40950	4.60
24	-100	0	0.15313	26304	2291	3677.11069	6256	1067	17.1	0.70864	4.63

PALOMAR - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	62.23758	26304	2291	1494511.00854	6256	1096	17.5	289.63392	4.65
2	-50	0	67.07392	26304	2291	1610646.04096	6256	1090	17.4	311.77817	4.65
3	-50	0	69.58692	26304	2291	1670990.70996	6256	1050	16.8	320.97401	4.61
4	50	0	76.6273	26304	2291	1840051.35490	6256	971	15.5	348.16487	4.54
5	50	0	101.35151	26304	2291	2433753.80963	6256	879	14.1	452.62299	4.47
6	50	0	132.881	26304	2291	3190871.45300	6256	788	12.6	583.55367	4.39
7	50	0	166.85749	26304	2291	4006748.90737	6256	701	11.2	721.28693	4.32
8	50	0	199.35655	26304	2291	4787148.83515	6256	628	10.0	850.59503	4.27
9	50	0	227.0465	26304	2291	5452067.60450	6256	679	10.9	977.59864	4.31
10	50	0	258.20597	26304	2291	6200299.95761	6256	589	9.4	1094.10622	4.24
11	50	0	284.95975	26304	2291	6842738.47675	6256	540	8.6	1197.12010	4.20
12	50	0	306.84919	26304	2291	7368369.59947	6256	518	8.3	1284.13552	4.18
13	50	0	305.48615	26304	2291	7335638.91995	6256	517	8.3	1278.20856	4.18
14	50	0	284.9321	26304	2291	6842074.51730	6256	523	8.4	1193.45448	4.19
15	50	0	255.29701	26304	2291	6130447.10113	6256	550	8.8	1074.38610	4.21
16	50	0	222.46841	26304	2291	5342133.92933	6256	596	9.5	943.83992	4.24
17	50	0	190.65477	26304	2291	4578192.99201	6256	516	8.2	797.59460	4.18
18	50	0	149.99496	26304	2291	3601828.97448	6256	612	9.8	638.16956	4.25
19	50	0	109.43689	26304	2291	2627908.03957	6256	701	11.2	473.07075	4.32
20	50	0	71.34752	26304	2291	1713267.99776	6256	802	12.8	314.13055	4.40
21	50	0	47.98635	26304	2291	1152296.22255	6256	895	14.3	214.94054	4.48
22	-50	50	46.33971	26304	2291	1112755.45623	6256	980	15.7	210.90892	4.55
23	-50	0	48.61618	26304	2291	1167420.33034	6256	1040	16.6	223.81525	4.60
24	-50	0	55.01306	26304	2291	1321028.60978	6256	1067	17.1	254.58250	4.63

POMONA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	300	-100	0.0378	26280	432	977.05440	6248	138	2.2	0.15991	4.23
2	200	-50	0.08941	26280	432	2311.06968	6248	140	2.2	0.37837	4.23
3	200	-50	0.18145	26280	432	4690.11960	6248	142	2.3	0.76812	4.23
4	200	-50	0.30538	26280	432	7893.46224	6248	145	2.3	1.29337	4.24
5	200	-50	0.4489	26280	432	11603.16720	6248	147	2.4	1.90185	4.24
6	200	0	0.59344	26280	432	15339.23712	6248	152	2.4	2.51628	4.24
7	200	0	0.72765	26280	432	18808.29720	6248	154	2.5	3.08636	4.24
8	250	0	0.84968	26280	432	21962.52864	6248	157	2.5	3.60573	4.24
9	250	0	0.93127	26280	432	24071.46696	6248	159	2.5	3.95327	4.25
10	250	0	0.9478	26280	432	24498.73440	6248	158	2.5	4.02278	4.24
11	250	0	0.89255	26280	432	23070.63240	6248	157	2.5	3.78766	4.24
12	250	0	0.7753	26280	432	20039.95440	6248	154	2.5	3.28847	4.24
13	300	0	0.63398	26280	432	16387.11504	6248	149	2.4	2.68685	4.24
14	300	0	0.49462	26280	432	12784.93776	6248	145	2.3	2.09486	4.24
15	300	50	0.35974	26280	432	9298.55952	6248	142	2.3	1.52286	4.23
16	350	50	0.22753	26280	432	5881.19544	6248	139	2.2	0.96271	4.23
17	350	50	0.11619	26280	432	3003.27912	6248	135	2.2	0.49129	4.23
18	400	0	0.03912	26280	432	1011.17376	6248	134	2.1	0.16539	4.23
19	0	-50	0.0042	26280	432	108.56160	6248	133	2.1	0.01775	4.23
20	0	-50	0.00468	26280	432	120.96864	6248	133	2.1	0.01978	4.23
21	0	-50	0.0052	26280	432	134.40960	6248	136	2.2	0.02199	4.23
22	0	-50	0.00567	26280	432	146.55816	6248	135	2.2	0.02397	4.23
23	0	-50	0.00623	26280	432	161.03304	6248	136	2.2	0.02635	4.23
24	500	-250	0.01616	26280	432	417.70368	6248	136	2.2	0.06834	4.23

POMONA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	0.59146	26280	432	15288.05808	6248	138	2.2	2.50214	4.23
2	100	0	1.20437	26280	432	31130.55576	6248	140	2.2	5.09669	4.23
3	100	0	2.08811	26280	432	53973.46728	6248	142	2.3	8.83941	4.23
4	100	0	3.14746	26280	432	81355.54608	6248	145	2.3	13.33042	4.24
5	100	0	4.34608	26280	432	112337.47584	6248	147	2.4	18.41296	4.24
6	100	0	5.57952	26280	432	144219.43296	6248	152	2.4	23.65804	4.24
7	100	0	6.79151	26280	432	175546.95048	6248	154	2.5	28.80652	4.24
8	100	0	7.82163	26280	432	202173.49224	6248	157	2.5	33.19217	4.24
9	100	0	8.41525	26280	432	217517.38200	6248	159	2.5	35.72301	4.25
10	100	0	8.44758	26280	432	218353.04784	6248	158	2.5	35.85436	4.24
11	100	0	7.8987	26280	432	204165.59760	6248	157	2.5	33.51922	4.24
12	100	0	6.84909	26280	432	177035.27832	6248	154	2.5	29.05075	4.24
13	100	0	5.65066	26280	432	146058.25968	6248	149	2.4	23.94790	4.24
14	100	0	4.41875	26280	432	114215.85000	6248	145	2.3	18.71471	4.24
15	100	0	3.20379	26280	432	82811.56392	6248	142	2.3	13.56233	4.23
16	150	0	2.10868	26280	432	54505.16064	6248	139	2.2	8.92211	4.23
17	150	0	1.168	26280	432	30190.46400	6248	135	2.2	4.93873	4.23
18	200	0	0.48016	26280	432	12411.17568	6248	134	2.1	2.02996	4.23
19	500	-200	0.19471	26280	432	5032.86408	6248	133	2.1	0.82304	4.23
20	500	0	0.07366	26280	432	1903.96368	6248	133	2.1	0.31136	4.23
21	0	-50	0.04644	26280	432	1200.38112	6248	136	2.2	0.19640	4.23
22	0	-50	0.05041	26280	432	1302.99768	6248	135	2.2	0.21315	4.23
23	0	-50	0.05369	26280	432	1387.77912	6248	136	2.2	0.22706	4.23
24	100	-50	0.21115	26280	432	5457.80520	6248	136	2.2	0.89297	4.23

POMONA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	65.9476	26280	432	1704613.56480	6248	138	2.2	278.98749	4.23
2	50	0	58.23568	26280	432	1505275.85664	6248	140	2.2	246.44333	4.23
3	50	0	70.24739	26280	432	1815754.53672	6248	142	2.3	297.37218	4.23
4	50	0	88.80241	26280	432	2295364.69368	6248	145	2.3	376.10432	4.24
5	50	0	111.03137	26280	432	2869938.85176	6248	147	2.4	470.40466	4.24
6	50	0	135.13711	26280	432	3493024.01928	6248	152	2.4	573.00263	4.24
7	50	0	158.47651	26280	432	4096300.83048	6248	154	2.5	672.18589	4.24
8	50	0	179.27428	26280	432	4633881.58944	6248	157	2.5	760.77517	4.24
9	50	0	197.23857	26280	432	5098222.55736	6248	159	2.5	837.28405	4.25
10	50	0	218.81575	26280	432	5655949.50600	6248	158	2.5	928.72734	4.24
11	50	0	244.03622	26280	432	6307848.21456	6248	157	2.5	1035.60141	4.24
12	50	0	270.93265	26280	432	7003067.13720	6248	154	2.5	1149.17413	4.24
13	50	0	285.34864	26280	432	7375691.64672	6248	149	2.4	1209.32803	4.24
14	50	0	285.77704	26280	432	7386764.92992	6248	145	2.3	1210.34982	4.24
15	50	0	275.07823	26280	432	7110222.08904	6248	142	2.3	1164.46480	4.23
16	50	0	256.69684	26280	432	6635099.92032	6248	139	2.2	1086.11883	4.23
17	50	0	236.76058	26280	432	6119787.47184	6248	135	2.2	1001.11033	4.23
18	50	0	207.98698	26280	432	5376047.45904	6248	134	2.1	879.30119	4.23
19	50	0	170.7548	26280	432	4413670.07040	6248	133	2.1	721.77761	4.23
20	100	-50	154.35448	26280	432	3989754.59904	6248	133	2.1	652.45374	4.23
21	100	-50	130.80712	26280	432	3381102.43776	6248	136	2.2	553.19084	4.23
22	100	-50	109.58201	26280	432	2832475.79448	6248	135	2.2	463.35282	4.23
23	100	-50	93.63298	26280	432	2420225.26704	6248	136	2.2	395.97926	4.23
24	100	-50	78.6095	26280	432	2031898.35600	6248	136	2.2	332.44410	4.23

REDLANDS - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-500	0	0.04181	8760	478	346.27042	2088	291	13.9	0.19269	4.61
2	150	-100	0.08511	8760	478	704.88102	2088	250	12.0	0.38350	4.51
3	150	-100	0.18241	8760	478	1510.71962	2088	209	10.0	0.80400	4.41
4	150	-100	0.31173	8760	478	2581.74786	2088	167	8.0	1.34396	4.31
5	150	-100	0.45602	8760	478	3776.75764	2088	125	6.0	1.92397	4.22
6	200	-100	0.60555	8760	478	5015.16510	2088	84	4.0	2.50258	4.13
7	200	-50	0.75634	8760	478	6264.00788	2088	51	2.4	3.07511	4.07
8	200	-100	0.88379	8760	478	7319.54878	2088	31	1.5	3.55836	4.03
9	200	-50	0.9679	8760	478	8016.14780	2088	25	1.2	3.88568	4.01
10	250	-50	0.99231	8760	478	8218.31142	2088	20	1.0	3.97404	4.00
11	250	-50	0.94769	8760	478	7848.76858	2088	20	1.0	3.79534	4.00
12	250	-50	0.83365	8760	478	6904.28930	2088	21	1.0	3.34025	4.01
13	250	-50	0.69935	8760	478	5792.01670	2088	35	1.7	2.82125	4.03
14	300	-50	0.54905	8760	478	4547.23210	2088	53	2.5	2.23451	4.07
15	300	-50	0.40803	8760	478	3379.30446	2088	83	4.0	1.68544	4.13
16	300	-50	0.27569	8760	478	2283.26458	2088	120	5.7	1.16020	4.21
17	350	-50	0.15386	8760	478	1274.26852	2088	162	7.8	0.66161	4.30
18	400	-50	0.05645	8760	478	467.51890	2088	208	10.0	0.24868	4.41
19	-50	0	0.00342	8760	478	28.32444	2088	249	11.9	0.01540	4.50
20	-50	0	0.00391	8760	478	32.38262	2088	290	13.9	0.01801	4.61
21	-50	0	0.0043	8760	478	35.61260	2088	318	15.2	0.02012	4.68
22	-50	0	0.0046	8760	478	38.09720	2088	341	16.3	0.02181	4.74
23	-50	0	0.00521	8760	478	43.14922	2088	344	16.5	0.02474	4.75
24	-500	50	0.01975	8760	478	163.56950	2088	327	15.7	0.09288	4.70

REDLANDS - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	WORKER PERIOD AVE CONC	% WORKER CALM & MISSING HRS	QUOTIENT (FACTOR)
1	-50	0	0.52894	8760	478	4380.68108	2088	291	2.43777	13.9	4.61
2	50	-50	1.22841	8760	478	10173.69162	2088	250	5.53520	12.0	4.51
3	50	-50	2.14057	8760	478	17728.20074	2088	209	9.43491	10.0	4.41
4	50	-50	3.12441	8760	478	25876.36362	2088	167	13.47026	8.0	4.31
5	100	-50	4.19282	8760	478	34724.93524	2088	125	17.68973	6.0	4.22
6	100	-50	5.31036	8760	478	43980.40152	2088	84	21.94631	4.0	4.13
7	100	-50	6.45196	8760	478	53435.13272	2088	51	26.23227	2.4	4.07
8	100	-50	7.43242	8760	478	61555.30244	2088	31	29.92479	1.5	4.03
9	100	-50	7.96745	8760	478	65986.42090	2088	25	31.98566	1.2	4.01
10	100	-50	7.90056	8760	478	65432.43792	2088	20	31.64044	1.0	4.00
11	100	-50	7.20298	8760	478	59655.08036	2088	20	28.84675	1.0	4.00
12	100	-50	6.14084	8760	478	50858.43688	2088	21	24.60495	1.0	4.01
13	100	0	5.07104	8760	478	41998.35328	2088	35	20.45706	1.7	4.03
14	150	-50	4.07763	8760	478	33770.93166	2088	53	16.59505	2.5	4.07
15	150	0	3.14168	8760	478	26019.39376	2088	83	12.97725	4.0	4.13
16	150	0	2.23696	8760	478	18526.50272	2088	120	9.41387	5.7	4.21
17	150	0	1.32077	8760	478	10938.61714	2088	162	5.67945	7.8	4.30
18	150	0	0.517	8760	478	4281.79400	2088	208	2.27755	10.0	4.41
19	500	-100	0.07352	8760	478	608.89264	2088	249	0.33110	11.9	4.50
20	-50	0	0.04779	8760	478	395.79678	2088	290	0.22013	13.9	4.61
21	-50	0	0.05202	8760	478	430.82964	2088	318	0.24341	15.2	4.68
22	-50	0	0.05512	8760	478	456.50384	2088	341	0.26131	16.3	4.74
23	-50	0	0.05897	8760	478	488.38954	2088	344	0.28004	16.5	4.75
24	-50	0	0.18742	8760	478	1552.21244	2088	327	0.88144	15.7	4.70

REDLANDS - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-300	50	45.47894	8760	478	376656.58108	2088	291	13.9	209.60299	4.61
2	-50	0	45.80464	8760	478	379354.02848	2088	250	12.0	206.39501	4.51
3	-50	0	53.94402	8760	478	446764.37364	2088	209	10.0	237.76710	4.41
4	50	0	74.29323	8760	478	615296.53086	2088	167	8.0	320.30012	4.31
5	50	0	96.44381	8760	478	798747.63442	2088	125	6.0	406.90149	4.22
6	50	0	123.94464	8760	478	1026509.50848	2088	84	4.0	512.23029	4.13
7	50	0	151.19332	8760	478	1252183.07624	2088	51	2.4	614.71923	4.07
8	50	0	175.86202	8760	478	1456489.24964	2088	31	1.5	708.06478	4.03
9	50	0	200.54185	8760	478	1660887.60170	2088	25	1.2	805.08367	4.01
10	50	0	230.43001	8760	478	1908421.34282	2088	20	1.0	922.83431	4.00
11	50	0	263.81094	8760	478	2184882.20508	2088	20	1.0	1056.51944	4.00
12	50	0	299.22627	8760	478	2478191.96814	2088	21	1.0	1198.93177	4.01
13	50	0	298.91289	8760	478	2475596.55498	2088	35	1.7	1205.84343	4.03
14	50	0	277.77399	8760	478	2300524.18518	2088	53	2.5	1130.47872	4.07
15	50	0	252.24911	8760	478	2089127.12902	2088	83	4.0	1041.95867	4.13
16	50	0	224.21967	8760	478	1856987.30694	2088	120	5.7	943.59111	4.21
17	50	0	190.84881	8760	478	1580609.84442	2088	162	7.8	820.66970	4.30
18	50	0	147.20039	8760	478	1219113.62998	2088	208	10.0	648.46470	4.41
19	50	0	96.70574	8760	478	800916.93868	2088	249	11.9	435.51764	4.50
20	100	-50	65.67926	8760	478	543955.63132	2088	290	13.9	302.53372	4.61
21	100	-50	44.74535	8760	478	370580.98870	2088	318	15.2	209.36779	4.68
22	-300	50	46.41385	8760	478	384399.50570	2088	341	16.3	220.03406	4.74
23	-300	50	48.26296	8760	478	399713.83472	2088	344	16.5	229.19371	4.75
24	-300	50	48.06504	8760	478	398074.66128	2088	327	15.7	226.05035	4.70

SAN BERNARDINO - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	350	0.04085	26280	1292	1020.75980	6248	872	14.0	0.18987	4.65
2	100	200	0.09946	26280	1292	2485.30648	6248	823	13.2	0.45812	4.61
3	100	150	0.20057	26280	1292	5011.84316	6248	744	11.9	0.91058	4.54
4	100	150	0.33332	26280	1292	8329.00016	6248	636	10.2	1.48414	4.45
5	150	150	0.48464	26280	1292	12110.18432	6248	526	8.4	2.11643	4.37
6	150	150	0.64456	26280	1292	16106.26528	6248	414	6.6	2.76076	4.28
7	150	150	0.79252	26280	1292	19803.48976	6248	312	5.0	3.33617	4.21
8	150	150	0.92034	26280	1292	22997.45592	6248	206	3.3	3.80627	4.14
9	200	200	1.02323	26280	1292	25568.47124	6248	138	2.2	4.18469	4.09
10	200	200	1.0794	26280	1292	26972.04720	6248	99	1.6	4.38641	4.06
11	200	200	1.04725	26280	1292	26168.68300	6248	87	1.4	4.24747	4.06
12	200	200	0.92541	26280	1292	23124.14508	6248	91	1.5	3.75575	4.06
13	200	200	0.78218	26280	1292	19545.11384	6248	92	1.5	3.17497	4.06
14	250	250	0.6348	26280	1292	15862.38240	6248	109	1.7	2.58387	4.07
15	250	250	0.49254	26280	1292	12307.58952	6248	150	2.4	2.01830	4.10
16	250	250	0.34312	26280	1292	8573.88256	6248	208	3.3	1.41952	4.14
17	300	300	0.19921	26280	1292	4977.85948	6248	282	4.5	0.83437	4.19
18	300	300	0.08024	26280	1292	2005.03712	6248	370	5.9	0.34111	4.25
19	500	500	0.0042	26280	1292	104.94960	6248	461	7.4	0.01814	4.32
20	500	-400	0.00275	26280	1292	68.71700	6248	565	9.0	0.01209	4.40
21	-50	0	0.00279	26280	1292	69.71652	6248	674	10.8	0.01251	4.48
22	-50	0	0.00305	26280	1292	76.21340	6248	769	12.3	0.01391	4.56
23	500	-450	0.00363	26280	1292	90.70644	6248	830	13.3	0.01674	4.61
24	500	-400	0.01549	26280	1292	387.06412	6248	878	14.1	0.07208	4.65

SAN BERNARDINO - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	0.61923	26280	1292	15473.31924	6248	872	14.0	2.87822	4.65
2	50	50	1.30694	26280	1292	32657.81672	6248	823	13.2	6.01987	4.61
3	50	50	2.2765	26280	1292	56885.18200	6248	744	11.9	10.33524	4.54
4	50	50	3.33493	26280	1292	83333.23084	6248	636	10.2	14.84911	4.45
5	50	50	4.37187	26280	1292	109244.28756	6248	526	8.4	19.09198	4.37
6	50	50	5.37512	26280	1292	134313.49856	6248	414	6.6	23.02254	4.28
7	50	100	6.31892	26280	1292	157897.17296	6248	312	5.0	26.59993	4.21
8	100	100	7.24372	26280	1292	181006.07536	6248	206	3.3	29.95797	4.14
9	100	100	8.1813	26280	1292	204434.32440	6248	138	2.2	33.45897	4.09
10	100	100	8.82249	26280	1292	220456.38012	6248	99	1.6	35.85240	4.06
11	100	100	8.99277	26280	1292	224711.33676	6248	87	1.4	36.47319	4.06
12	100	100	8.30546	26280	1292	207536.83448	6248	91	1.5	33.70746	4.06
13	100	100	7.26975	26280	1292	181656.51300	6248	92	1.5	29.50886	4.06
14	100	100	6.13035	26280	1292	153185.18580	6248	109	1.7	24.95279	4.07
15	100	100	4.96832	26280	1292	124148.38016	6248	150	2.4	20.35887	4.10
16	100	100	3.72613	26280	1292	93108.53644	6248	208	3.3	15.41532	4.14
17	100	100	2.45722	26280	1292	61401.01336	6248	282	4.5	10.29182	4.19
18	150	150	1.45646	26280	1292	36394.02248	6248	370	5.9	6.19157	4.25
19	250	300	0.78676	26280	1292	19659.55888	6248	461	7.4	3.39719	4.32
20	400	500	0.34453	26280	1292	8609.11564	6248	565	9.0	1.51489	4.40
21	400	500	0.1543	26280	1292	3855.64840	6248	674	10.8	0.69172	4.48
22	150	-100	0.09964	26280	1292	2489.80432	6248	769	12.3	0.45443	4.56
23	150	-100	0.1332	26280	1292	3328.40160	6248	830	13.3	0.61432	4.61
24	150	-100	0.22779	26280	1292	5692.01652	6248	878	14.1	1.05997	4.65

SAN BERNARDINO - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	63.46595	26280	1292	1585887.15860	6248	872	14.0	294.99389	4.65
2	0	50	55.96467	26280	1292	1398445.17396	6248	823	13.2	257.77791	4.61
3	0	50	65.81835	26280	1292	1644668.92980	6248	744	11.9	298.81340	4.54
4	0	50	76.94855	26280	1292	1922790.36740	6248	636	10.2	342.62123	4.45
5	0	50	88.11255	26280	1292	2201756.39940	6248	526	8.4	384.78791	4.37
6	0	50	98.59945	26280	1292	2463803.05660	6248	414	6.6	422.31797	4.28
7	0	50	107.32754	26280	1292	2681900.56952	6248	312	5.0	451.80266	4.21
8	0	50	112.73519	26280	1292	2817026.92772	6248	206	3.3	466.24080	4.14
9	50	50	120.54293	26280	1292	3012126.73484	6248	138	2.2	492.98310	4.09
10	50	50	141.77071	26280	1292	3542566.50148	6248	99	1.6	576.12075	4.06
11	50	50	169.40463	26280	1292	4233082.89444	6248	87	1.4	687.07724	4.06
12	50	50	207.02118	26280	1292	5173045.24584	6248	91	1.5	840.18926	4.06
13	50	50	237.14305	26280	1292	5925730.53340	6248	92	1.5	962.59430	4.06
14	50	50	260.28953	26280	1292	6504114.77564	6248	109	1.7	1059.47463	4.07
15	50	50	274.82077	26280	1292	6867221.40076	6248	150	2.4	1126.14323	4.10
16	50	50	274.32052	26280	1292	6854721.15376	6248	208	3.3	1134.88761	4.14
17	50	50	267.24594	26280	1292	6677941.54872	6248	282	4.5	1119.33315	4.19
18	50	50	247.00929	26280	1292	6172268.13852	6248	370	5.9	1050.06263	4.25
19	50	50	216.76584	26280	1292	5416544.80992	6248	461	7.4	935.98493	4.32
20	50	100	173.1904	26280	1292	4327681.71520	6248	565	9.0	761.51359	4.40
21	50	100	149.39248	26280	1292	3733019.29024	6248	674	10.8	669.72000	4.48
22	50	100	121.76981	26280	1292	3042784.01228	6248	769	12.3	555.35390	4.56
23	50	100	100.07427	26280	1292	2500655.85876	6248	830	13.3	461.54593	4.61
24	50	100	79.55709	26280	1292	1987972.56492	6248	878	14.1	370.19973	4.65

KEARNY MESA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-150	0.08297	26304	1813	2032.01827	7830	910	11.6	0.29364	3.54
2	150	-100	0.15998	26304	1813	3918.07018	7830	907	11.6	0.56595	3.54
3	200	-100	0.26694	26304	1813	6537.62754	7830	886	11.3	0.94148	3.53
4	200	-100	0.38512	26304	1813	9431.97392	7830	872	11.1	1.35556	3.52
5	200	-100	0.50152	26304	1813	12282.72632	7830	856	10.9	1.76122	3.51
6	200	-100	0.61064	26304	1813	14955.18424	7830	848	10.8	2.14196	3.51
7	200	-100	0.69021	26304	1813	16903.93311	7830	849	10.8	2.42142	3.51
8	250	-100	0.73932	26304	1813	18106.68612	7830	817	10.4	2.58187	3.49
9	250	-100	0.75042	26304	1813	18378.53622	7830	755	9.6	2.59767	3.46
10	250	-100	0.72932	26304	1813	17861.77612	7830	685	8.7	2.49990	3.43
11	250	-100	0.68371	26304	1813	16744.74161	7830	645	8.2	2.33051	3.41
12	250	-100	0.60961	26304	1813	14929.95851	7830	621	7.9	2.07102	3.40
13	250	-100	0.50731	26304	1813	12424.52921	7830	610	7.8	1.72085	3.39
14	250	-100	0.38994	26304	1813	9550.02054	7830	593	7.6	1.31961	3.38
15	300	-150	0.27924	26304	1813	6838.86684	7830	590	7.5	0.94459	3.38
16	300	-150	0.16786	26304	1813	4111.05926	7830	592	7.6	0.56798	3.38
17	300	-150	0.07795	26304	1813	1909.07345	7830	606	7.7	0.26427	3.39
18	350	-200	0.02278	26304	1813	557.90498	7830	645	8.2	0.07765	3.41
19	0	500	0.00482	26304	1813	118.04662	7830	702	9.0	0.01656	3.44
20	0	500	0.00483	26304	1813	118.29153	7830	762	9.7	0.01674	3.47
21	0	500	0.00496	26304	1813	121.47536	7830	797	10.2	0.01727	3.48
22	-50	500	0.00874	26304	1813	214.05134	7830	825	10.5	0.03056	3.50
23	-50	500	0.02154	26304	1813	527.53614	7830	859	11.0	0.07568	3.51
24	0	300	0.04544	26304	1813	1112.87104	7830	898	11.5	0.16054	3.53

KEARNY MESA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.35817	26304	1813	33262.94147	7830	910	11.6	4.80678	3.54
2	50	50	2.11813	26304	1813	51875.12183	7830	907	11.6	7.49316	3.54
3	50	50	2.81323	26304	1813	68898.81593	7830	886	11.3	9.92206	3.53
4	50	50	3.40099	26304	1813	83293.64609	7830	872	11.1	11.97092	3.52
5	100	-50	4.27704	26304	1813	104748.98664	7830	856	10.9	15.01993	3.51
6	100	-50	5.2404	26304	1813	128342.63640	7830	848	10.8	18.38193	3.51
7	100	-50	6.03015	26304	1813	147684.40365	7830	849	10.8	21.15519	3.51
8	100	-50	6.5101	26304	1813	159438.85910	7830	817	10.4	22.73476	3.49
9	100	-50	6.57622	26304	1813	161058.20402	7830	755	9.6	22.76441	3.46
10	100	-50	6.3076	26304	1813	154479.43160	7830	685	8.7	21.62063	3.43
11	100	-50	5.84464	26304	1813	143141.07824	7830	645	8.2	19.92221	3.41
12	100	-50	5.22149	26304	1813	127879.51159	7830	621	7.9	17.73887	3.40
13	100	-50	4.43399	26304	1813	108592.84909	7830	610	7.8	15.04056	3.39
14	100	-50	3.50471	26304	1813	85833.85261	7830	593	7.6	11.86042	3.38
15	100	-50	2.50936	26304	1813	61456.73576	7830	590	7.5	8.48850	3.38
16	100	-50	1.54547	26304	1813	37850.10577	7830	592	7.6	5.22936	3.38
17	150	-50	0.78926	26304	1813	19329.76666	7830	606	7.7	2.67577	3.39
18	200	-100	0.30774	26304	1813	7536.86034	7830	645	8.2	1.04897	3.41
19	0	150	0.18342	26304	1813	4492.13922	7830	702	9.0	0.63021	3.44
20	0	150	0.16993	26304	1813	4161.75563	7830	762	9.7	0.58882	3.47
21	0	150	0.16545	26304	1813	4052.03595	7830	797	10.2	0.57615	3.48
22	0	150	0.21125	26304	1813	5173.72375	7830	825	10.5	0.73858	3.50
23	0	100	0.41536	26304	1813	10172.58176	7830	859	11.0	1.45927	3.51
24	0	100	0.83705	26304	1813	20500.19155	7830	898	11.5	2.95733	3.53

KEARNY MESA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	68.76835	26304	1813	1684205.65985	7830	910	11.6	243.38232	3.54
2	0	50	74.07187	26304	1813	1814094.16817	7830	907	11.6	262.03874	3.54
3	0	50	78.4778	26304	1813	1921999.79980	7830	886	11.3	276.78569	3.53
4	50	0	81.98311	26304	1813	2007848.34701	7830	872	11.1	288.56688	3.52
5	50	0	99.45639	26304	1813	2435786.44749	7830	856	10.9	349.26677	3.51
6	50	0	117.63254	26304	1813	2880938.53714	7830	848	10.8	412.62368	3.51
7	50	0	134.71148	26304	1813	3299218.85668	7830	849	10.8	472.59975	3.51
8	50	0	151.26253	26304	1813	3704570.62223	7830	817	10.4	528.24335	3.49
9	50	0	164.57775	26304	1813	4030673.67525	7830	755	9.6	569.70653	3.46
10	50	0	175.05832	26304	1813	4287353.31512	7830	685	8.7	600.04945	3.43
11	50	0	176.15086	26304	1813	4314110.71226	7830	645	8.2	600.43295	3.41
12	50	0	169.94269	26304	1813	4162066.42079	7830	621	7.9	577.34310	3.40
13	50	0	158.91434	26304	1813	3891971.10094	7830	610	7.8	539.05417	3.39
14	50	0	144.4592	26304	1813	3537950.26720	7830	593	7.6	488.86973	3.38
15	50	-50	129.79889	26304	1813	3178904.61499	7830	590	7.5	439.07522	3.38
16	50	-50	127.14583	26304	1813	3113928.52253	7830	592	7.6	430.21947	3.38
17	50	-50	122.72119	26304	1813	3005564.66429	7830	606	7.7	416.05269	3.39
18	50	-50	111.89165	26304	1813	2740338.40015	7830	645	8.2	381.39713	3.41
19	50	-50	97.37192	26304	1813	2384735.69272	7830	702	9.0	334.55888	3.44
20	50	-50	76.25987	26304	1813	1867680.47617	7830	762	9.7	264.24455	3.47
21	0	50	59.92054	26304	1813	1467513.94514	7830	797	10.2	208.66116	3.48
22	0	50	56.81233	26304	1813	1391390.77403	7830	825	10.5	198.62823	3.50
23	0	50	58.33987	26304	1813	1428801.75617	7830	859	11.0	204.96367	3.51
24	0	50	63.14546	26304	1813	1546495.46086	7830	898	11.5	223.09513	3.53

PALOMAR - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	50	0.11461	26304	2291	2752.12993	7820	1313	16.8	0.42295	3.69
2	150	50	0.21952	26304	2291	5271.33376	7820	1235	15.8	0.80051	3.65
3	200	50	0.34291	26304	2291	8234.29783	7820	1156	14.8	1.23564	3.60
4	200	50	0.47006	26304	2291	11287.55078	7820	1071	13.7	1.67248	3.56
5	200	0	0.59099	26304	2291	14191.44287	7820	985	12.6	2.07629	3.51
6	200	0	0.70014	26304	2291	16812.46182	7820	902	11.5	2.43025	3.47
7	250	0	0.78328	26304	2291	18808.90264	7820	951	12.2	2.73823	3.50
8	250	0	0.83593	26304	2291	20073.18709	7820	858	11.0	2.88325	3.45
9	250	0	0.84409	26304	2291	20269.13317	7820	757	9.7	2.86976	3.40
10	250	0	0.8161	26304	2291	19597.00930	7820	663	8.5	2.73816	3.36
11	250	0	0.75885	26304	2291	18222.26505	7820	623	8.0	2.53193	3.34
12	250	0	0.66899	26304	2291	16064.45687	7820	623	8.0	2.23210	3.34
13	250	0	0.54882	26304	2291	13178.81466	7820	656	8.4	1.83959	3.35
14	250	0	0.41206	26304	2291	9894.79678	7820	710	9.1	1.39167	3.38
15	300	0	0.27978	26304	2291	6718.35714	7820	766	9.8	0.95242	3.40
16	300	-50	0.16245	26304	2291	3900.91185	7820	842	10.8	0.55903	3.44
17	300	-100	0.08094	26304	2291	1943.61222	7820	779	10.0	0.27604	3.41
18	300	-150	0.02496	26304	2291	599.36448	7820	876	11.2	0.08631	3.46
19	-450	-200	0.00494	26304	2291	118.62422	7820	978	12.5	0.01734	3.51
20	-400	-150	0.00466	26304	2291	111.90058	7820	1085	13.9	0.01661	3.57
21	-400	-200	0.00408	26304	2291	97.97304	7820	1179	15.1	0.01475	3.62
22	-500	-250	0.00734	26304	2291	176.25542	7820	1254	16.0	0.02684	3.66
23	-50	250	0.01896	26304	2291	455.28648	7820	1312	16.8	0.06996	3.69
24	100	150	0.05053	26304	2291	1213.37689	7820	1336	17.1	0.18713	3.70

PALOMAR - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.79401	26304	2291	43079.56213	7820	1313	16.8	6.62050	3.69
2	50	50	2.7745	26304	2291	66624.06850	7820	1235	15.8	10.11755	3.65
3	100	0	4.02097	26304	2291	96555.55261	7820	1156	14.8	14.48913	3.60
4	100	0	5.71297	26304	2291	137185.54861	7820	1071	13.7	20.32680	3.56
5	100	0	7.47105	26304	2291	179402.32365	7820	985	12.6	26.24760	3.51
6	100	0	9.08402	26304	2291	218134.57226	7820	902	11.5	31.53145	3.47
7	100	0	10.25315	26304	2291	246208.89095	7820	951	12.2	35.84348	3.50
8	100	0	10.98429	26304	2291	263765.75577	7820	858	11.0	37.88649	3.45
9	100	0	11.11226	26304	2291	266838.69938	7820	757	9.7	37.77980	3.40
10	100	0	10.70486	26304	2291	257055.80318	7820	663	8.5	35.91670	3.36
11	100	0	9.8762	26304	2291	237157.19060	7820	623	8.0	32.95223	3.34
12	100	0	8.79903	26304	2291	211291.10739	7820	623	8.0	29.35822	3.34
13	100	0	7.34081	26304	2291	176274.87053	7820	656	8.4	24.60565	3.35
14	100	0	5.64239	26304	2291	135490.71107	7820	710	9.1	19.05636	3.38
15	100	0	3.89019	26304	2291	93415.13247	7820	766	9.8	13.24286	3.40
16	100	0	2.28302	26304	2291	54822.15926	7820	842	10.8	7.85643	3.44
17	150	0	1.19218	26304	2291	28627.81834	7820	779	10.0	4.06587	3.41
18	150	0	0.42743	26304	2291	10263.87659	7820	876	11.2	1.47809	3.46
19	500	100	0.13519	26304	2291	3246.31747	7820	978	12.5	0.47447	3.51
20	-100	-50	0.11603	26304	2291	2786.22839	7820	1085	13.9	0.41369	3.57
21	-100	-50	0.1019	26304	2291	2446.92470	7820	1179	15.1	0.36846	3.62
22	-100	0	0.13253	26304	2291	3182.44289	7820	1254	16.0	0.48469	3.66
23	-50	50	0.32155	26304	2291	7721.38015	7820	1312	16.8	1.18644	3.69
24	50	50	0.91054	26304	2291	21864.79702	7820	1336	17.1	3.37212	3.70

PALOMAR - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	64.60191	26304	2291	1551285.66483	7820	1313	16.8	238.40259	3.69
2	50	0	67.16566	26304	2291	1612848.99358	7820	1235	15.8	244.92771	3.65
3	50	0	86.7754	26304	2291	2083737.68020	7820	1156	14.8	312.68573	3.60
4	50	0	111.35187	26304	2291	2673892.45431	7820	1071	13.7	396.19091	3.56
5	50	0	139.09175	26304	2291	3340010.19275	7820	985	12.6	488.66279	3.51
6	50	0	167.58523	26304	2291	4024224.12799	7820	902	11.5	581.70340	3.47
7	50	0	194.22411	26304	2291	4663903.55343	7820	951	12.2	678.97853	3.50
8	50	0	224.85236	26304	2291	5399379.72068	7820	858	11.0	775.55009	3.45
9	50	0	252.42285	26304	2291	6061429.89705	7820	757	9.7	858.19480	3.40
10	50	0	275.34655	26304	2291	6611896.70515	7820	663	8.5	923.83634	3.36
11	50	0	282.82242	26304	2291	6791414.77146	7820	623	8.0	943.64524	3.34
12	50	0	277.9957	26304	2291	6675510.74410	7820	623	8.0	927.54075	3.34
13	50	0	262.24815	26304	2291	6297364.82595	7820	656	8.4	879.02915	3.35
14	50	0	239.25516	26304	2291	5745234.15708	7820	710	9.1	808.04981	3.38
15	50	0	213.26193	26304	2291	5121058.72509	7820	766	9.8	725.97941	3.40
16	50	0	185.3631	26304	2291	4451124.12030	7820	842	10.8	637.87964	3.44
17	50	0	158.33517	26304	2291	3802102.43721	7820	779	10.0	539.99467	3.41
18	50	0	125.85979	26304	2291	3022271.13727	7820	876	11.2	435.23490	3.46
19	50	0	93.2437	26304	2291	2239060.96810	7820	978	12.5	327.25241	3.51
20	50	0	62.12509	26304	2291	1491809.78617	7820	1085	13.9	221.50108	3.57
21	-50	0	47.17899	26304	2291	1132909.08687	7820	1179	15.1	170.59315	3.62
22	-50	0	51.9114	26304	2291	1246548.44820	7820	1254	16.0	189.84899	3.66
23	-50	0	57.95502	26304	2291	1391673.89526	7820	1312	16.8	213.84049	3.69
24	-50	0	62.2143	26304	2291	1493951.98590	7820	1336	17.1	230.40592	3.70

POMONA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	0	1.67498	26280	432	43294.88304	7810	175	2.2	5.67058	3.39
2	100	0	2.52254	26280	432	65202.61392	7810	179	2.3	8.54444	3.39
3	100	0	3.48087	26280	432	89973.52776	7810	183	2.3	11.79671	3.39
4	100	0	4.46874	26280	432	115507.99152	7810	188	2.4	15.15455	3.39
5	100	0	5.44049	26280	432	140625.78552	7810	189	2.4	18.45241	3.39
6	100	0	6.37933	26280	432	164892.92184	7810	192	2.5	21.64517	3.39
7	100	0	7.16963	26280	432	185320.59624	7810	193	2.5	24.32987	3.39
8	100	0	7.58985	26280	432	196182.44280	7810	193	2.5	25.75587	3.39
9	100	0	7.54073	26280	432	194912.78904	7810	194	2.5	25.59254	3.39
10	100	0	7.03831	26280	432	181926.23688	7810	193	2.5	23.88424	3.39
11	100	0	6.33091	26280	432	163641.36168	7810	190	2.4	21.47524	3.39
12	100	0	5.48577	26280	432	141796.18296	7810	188	2.4	18.60354	3.39
13	100	0	4.52666	26280	432	117005.10768	7810	184	2.4	15.34292	3.39
14	100	0	3.53869	26280	432	91468.05912	7810	179	2.3	11.98638	3.39
15	100	0	2.56683	26280	432	66347.42184	7810	174	2.2	8.68877	3.39
16	150	0	1.68973	26280	432	43676.14104	7810	170	2.2	5.71677	3.38
17	150	0	0.93943	26280	432	24282.38664	7810	168	2.2	3.17749	3.38
18	200	0	0.38972	26280	432	10073.48256	7810	168	2.2	1.31817	3.38
19	500	-200	0.15933	26280	432	4118.36184	7810	169	2.2	0.53898	3.38
20	500	0	0.06427	26280	432	1661.25096	7810	169	2.2	0.21741	3.38
21	0	-50	0.04922	26280	432	1272.23856	7810	171	2.2	0.16655	3.38
22	100	-50	0.17372	26280	432	4490.31456	7810	170	2.2	0.58774	3.38
23	100	-50	0.47768	26280	432	12347.07264	7810	170	2.2	1.61611	3.38
24	100	0	0.96732	26280	432	25003.28736	7810	171	2.2	3.27311	3.38

POMONA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	-50	0.14539	26280	432	3758.04072	7810	175	2.2	0.49221	3.39
2	200	-50	0.24454	26280	432	6320.86992	7810	179	2.3	0.82831	3.39
3	200	-50	0.35936	26280	432	9288.73728	7810	183	2.3	1.21788	3.39
4	200	0	0.475	26280	432	12277.80000	7810	188	2.4	1.61084	3.39
5	200	0	0.58245	26280	432	15055.16760	7810	189	2.4	1.97548	3.39
6	250	0	0.68649	26280	432	17744.39352	7810	192	2.5	2.32927	3.39
7	250	0	0.77125	26280	432	19935.27000	7810	193	2.5	2.61721	3.39
8	250	0	0.81936	26280	432	21178.81728	7810	193	2.5	2.78047	3.39
9	250	0	0.82376	26280	432	21292.54848	7810	194	2.5	2.79577	3.39
10	250	0	0.78241	26280	432	20223.73368	7810	193	2.5	2.65508	3.39
11	250	0	0.7142	26280	432	18460.64160	7810	190	2.4	2.42266	3.39
12	250	0	0.62035	26280	432	16034.80680	7810	188	2.4	2.10375	3.39
13	300	0	0.50729	26280	432	13112.43192	7810	184	2.4	1.71944	3.39
14	300	0	0.39583	26280	432	10231.41384	7810	179	2.3	1.34077	3.39
15	300	50	0.28793	26280	432	7442.41464	7810	174	2.2	0.97465	3.39
16	350	50	0.18215	26280	432	4708.21320	7810	170	2.2	0.61626	3.38
17	350	50	0.09308	26280	432	2405.93184	7810	168	2.2	0.31483	3.38
18	400	0	0.03142	26280	432	812.14416	7810	168	2.2	0.10627	3.38
19	0	-50	0.00464	26280	432	119.93472	7810	169	2.2	0.01570	3.38
20	0	-50	0.00508	26280	432	131.30784	7810	169	2.2	0.01718	3.38
21	0	-50	0.00569	26280	432	147.07512	7810	171	2.2	0.01925	3.38
22	500	-250	0.01302	26280	432	336.54096	7810	170	2.2	0.04405	3.38
23	300	-100	0.0304	26280	432	785.77920	7810	170	2.2	0.10285	3.38
24	200	-50	0.07176	26280	432	1854.85248	7810	171	2.2	0.24281	3.38

POMONA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKE HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	0	66.88293	26280	432	1728789.97464	7810	175	2.2	226.42960	3.39
2	50	0	78.93616	26280	432	2040341.86368	7810	179	2.3	267.37542	3.39
3	50	0	94.94525	26280	432	2454144.82200	7810	183	2.3	321.77066	3.39
4	50	0	113.62804	26280	432	2937057.57792	7810	188	2.4	385.33949	3.39
5	50	0	133.76259	26280	432	3457495.42632	7810	189	2.4	453.68002	3.39
6	50	0	155.21512	26280	432	4012000.42176	7810	192	2.5	526.64747	3.39
7	50	0	174.83572	26280	432	4519153.69056	7810	193	2.5	593.29837	3.39
8	50	0	196.43289	26280	432	5077397.34072	7810	193	2.5	666.58755	3.39
9	50	0	221.2805	26280	432	5719658.36400	7810	194	2.5	751.00556	3.39
10	50	0	249.09373	26280	432	6438574.73304	7810	193	2.5	845.29011	3.39
11	50	0	267.02625	26280	432	6902094.51000	7810	190	2.4	905.78668	3.39
12	50	0	271.20773	26280	432	7010177.40504	7810	188	2.4	919.72939	3.39
13	50	0	265.00007	26280	432	6849721.80936	7810	184	2.4	898.20637	3.39
14	50	0	252.4629	26280	432	6525661.03920	7810	179	2.3	855.15149	3.39
15	50	0	237.46298	26280	432	6137943.10704	7810	174	2.2	803.81654	3.39
16	50	0	219.40304	26280	432	5671129.77792	7810	170	2.2	742.29447	3.38
17	50	0	200.09348	26280	432	5172016.27104	7810	168	2.2	676.78831	3.38
18	50	0	174.28381	26280	432	4504887.92088	7810	168	2.2	589.49070	3.38
19	100	-50	148.72624	26280	432	3844275.85152	7810	169	2.2	503.11162	3.38
20	100	-50	136.06151	26280	432	3516917.91048	7810	169	2.2	460.26932	3.38
21	100	-50	116.42089	26280	432	3009247.16472	7810	171	2.2	393.93208	3.38
22	100	-50	95.89973	26280	432	2478816.22104	7810	170	2.2	324.45238	3.38
23	100	-50	79.98215	26280	432	2067378.61320	7810	170	2.2	270.59929	3.38
24	100	-50	67.81091	26280	432	1752776.40168	7810	171	2.2	229.45103	3.38

REDLANDS - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-100	0.14613	8760	478	1210.24866	2610	303	11.6	0.52460	3.59
2	150	-100	0.24958	8760	478	2067.02156	2610	258	9.9	0.87884	3.52
3	150	-100	0.36502	8760	478	3023.09564	2610	216	8.3	1.26278	3.46
4	200	-100	0.4846	8760	478	4013.45720	2610	172	6.6	1.64621	3.40
5	200	-50	0.6053	8760	478	5013.09460	2610	128	4.9	2.01978	3.34
6	200	-100	0.71152	8760	478	5892.80864	2610	86	3.3	2.33471	3.28
7	200	-50	0.79696	8760	478	6600.42272	2610	54	2.1	2.58233	3.24
8	250	-50	0.85358	8760	478	7069.34956	2610	36	1.4	2.74645	3.22
9	250	-50	0.87022	8760	478	7207.16204	2610	32	1.2	2.79564	3.21
10	250	-50	0.82892	8760	478	6865.11544	2610	29	1.1	2.65987	3.21
11	250	-50	0.75826	8760	478	6279.90932	2610	42	1.6	2.44545	3.23
12	250	-50	0.66701	8760	478	5524.17682	2610	58	2.2	2.16465	3.25
13	250	-50	0.55959	8760	478	4634.52438	2610	86	3.3	1.83618	3.28
14	300	-50	0.43933	8760	478	3638.53106	2610	122	4.7	1.46243	3.33
15	300	-50	0.32652	8760	478	2704.23864	2610	165	6.3	1.10603	3.39
16	300	-50	0.22066	8760	478	1827.50612	2610	213	8.2	0.76241	3.46
17	350	-50	0.12319	8760	478	1020.25958	2610	256	9.8	0.43342	3.52
18	400	-50	0.04524	8760	478	374.67768	2610	299	11.5	0.16213	3.58
19	-50	0	0.0038	8760	478	31.47160	2610	340	13.0	0.01386	3.65
20	-50	0	0.00417	8760	478	34.53594	2610	378	14.5	0.01547	3.71
21	-50	0	0.00479	8760	478	39.67078	2610	395	15.1	0.01791	3.74
22	-500	50	0.01591	8760	478	131.76662	2610	396	15.2	0.05952	3.74
23	-500	0	0.03356	8760	478	277.94392	2610	373	14.3	0.12425	3.70
24	150	-100	0.06827	8760	478	565.41214	2610	343	13.1	0.24941	3.65

REDLANDS - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	-50	1.71658	8760	478	14216.71556	2610	303	11.6	6.16243	3.59
2	50	-50	2.50366	8760	478	20735.31212	2610	258	9.9	8.81603	3.52
3	100	-50	3.35706	8760	478	27803.17092	2610	216	8.3	11.61369	3.46
4	100	-50	4.25095	8760	478	35206.36790	2610	172	6.6	14.44068	3.40
5	100	-50	5.1653	8760	478	42779.01460	2610	128	4.9	17.23570	3.34
6	100	-50	6.01292	8760	478	49799.00344	2610	86	3.3	19.73019	3.28
7	100	-50	6.72041	8760	478	55658.43562	2610	54	2.1	21.77560	3.24
8	100	-50	7.11772	8760	478	58948.95704	2610	36	1.4	22.90169	3.22
9	100	-50	7.01506	8760	478	58098.72692	2610	32	1.2	22.53636	3.21
10	100	-50	6.50262	8760	478	53854.69884	2610	29	1.1	20.86583	3.21
11	100	-50	5.76643	8760	478	47757.57326	2610	42	1.6	18.59719	3.23
12	100	-50	4.91534	8760	478	40708.84588	2610	58	2.2	15.95174	3.25
13	100	0	4.05934	8760	478	33619.45388	2610	86	3.3	13.31991	3.28
14	150	-50	3.26436	8760	478	27035.42952	2610	122	4.7	10.86633	3.33
15	150	0	2.51516	8760	478	20830.55512	2610	165	6.3	8.51965	3.39
16	150	0	1.79145	8760	478	14836.78890	2610	213	8.2	6.18973	3.46
17	150	0	1.05852	8760	478	8766.66264	2610	256	9.8	3.72416	3.52
18	150	0	0.41545	8760	478	3440.75690	2610	299	11.5	1.48886	3.58
19	500	-100	0.05953	8760	478	493.02746	2610	340	13.0	0.21719	3.65
20	-50	0	0.05022	8760	478	415.92204	2610	378	14.5	0.18635	3.71
21	-50	0	0.05482	8760	478	454.01924	2610	395	15.1	0.20497	3.74
22	-50	0	0.15882	8760	478	1315.34724	2610	396	15.2	0.59410	3.74
23	-50	0	0.43321	8760	478	3587.84522	2610	373	14.3	1.60386	3.70
24	50	-50	0.98664	8760	478	8171.35248	2610	343	13.1	3.60448	3.65

REDLANDS - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	45.3508	8760	478	375595.32560	2610	303	11.6	162.80682	3.59
2	50	0	60.52773	8760	478	501290.65986	2610	258	9.9	213.13378	3.52
3	50	0	78.2791	8760	478	648307.50620	2610	216	8.3	270.80514	3.46
4	50	0	100.35242	8760	478	831118.74244	2610	172	6.6	340.90186	3.40
5	50	0	123.30279	8760	478	1021193.70678	2610	128	4.9	411.43985	3.34
6	50	0	147.25117	8760	478	1219534.18994	2610	86	3.3	483.17519	3.28
7	50	0	173.53484	8760	478	1437215.54488	2610	54	2.1	562.29090	3.24
8	50	0	204.41071	8760	478	1692929.50022	2610	36	1.4	657.70377	3.22
9	50	0	237.08429	8760	478	1963532.08978	2610	32	1.2	761.64938	3.21
10	50	0	270.99063	8760	478	2244344.39766	2610	29	1.1	869.56389	3.21
11	50	0	274.80034	8760	478	2275896.41588	2610	42	1.6	886.25250	3.23
12	50	0	263.13703	8760	478	2179300.88246	2610	58	2.2	853.95803	3.25
13	50	0	247.94703	8760	478	2053497.30246	2610	86	3.3	813.58847	3.28
14	50	0	227.47119	8760	478	1883916.39558	2610	122	4.7	757.20112	3.33
15	50	0	205.25923	8760	478	1699956.94286	2610	165	6.3	695.27891	3.39
16	50	0	181.48141	8760	478	1503029.03762	2610	213	8.2	627.04591	3.46
17	50	0	154.0154	8760	478	1275555.54280	2610	256	9.8	541.86727	3.52
18	50	0	118.85346	8760	478	984344.35572	2610	299	11.5	425.93871	3.58
19	50	0	78.48865	8760	478	650042.99930	2610	340	13.0	286.36255	3.65
20	100	-50	55.02469	8760	478	455714.48258	2610	378	14.5	204.17316	3.71
21	-300	50	46.19985	8760	478	382627.15770	2610	395	15.1	172.74364	3.74
22	-300	50	45.56241	8760	478	377347.87962	2610	396	15.2	170.43716	3.74
23	-300	50	43.32203	8760	478	358793.05246	2610	373	14.3	160.39028	3.70
24	-300	50	40.49639	8760	478	335391.10198	2610	343	13.1	147.94491	3.65

SAN BERNARDINO - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	150	0.16062	26280	2291	3853.11318	7810	945	12.1	0.56127	3.49
2	100	150	0.26681	26280	2291	6400.50509	7810	857	11.0	0.92054	3.45
3	150	150	0.38784	26280	2291	9303.89376	7810	768	9.8	1.32120	3.41
4	150	150	0.51578	26280	2291	12373.04642	7810	659	8.4	1.73025	3.35
5	150	150	0.63431	26280	2291	15216.46259	7810	547	7.0	2.09507	3.30
6	150	150	0.74255	26280	2291	17813.03195	7810	433	5.5	2.41467	3.25
7	200	200	0.84805	26280	2291	20343.87145	7810	332	4.3	2.72050	3.21
8	200	200	0.92818	26280	2291	22266.11002	7810	229	2.9	2.93709	3.16
9	200	200	0.95389	26280	2291	22882.86721	7810	160	2.0	2.99122	3.14
10	200	200	0.91165	26280	2291	21869.57185	7810	125	1.6	2.84575	3.12
11	200	200	0.83833	26280	2291	20110.69837	7810	116	1.5	2.61382	3.12
12	200	200	0.74042	26280	2291	17761.93538	7810	132	1.7	2.31335	3.12
13	200	200	0.6259	26280	2291	15014.71510	7810	171	2.2	1.96553	3.14
14	250	250	0.50812	26280	2291	12189.29068	7810	227	2.9	1.60745	3.16
15	250	250	0.39411	26280	2291	9454.30479	7810	302	3.9	1.25923	3.20
16	250	250	0.27457	26280	2291	6586.65973	7810	393	5.0	0.88805	3.23
17	300	300	0.15944	26280	2291	3824.80616	7810	483	6.2	0.52202	3.27
18	300	300	0.06426	26280	2291	1541.53314	7810	591	7.6	0.21354	3.32
19	500	500	0.00341	26280	2291	81.80249	7810	703	9.0	0.01151	3.38
20	-50	0	0.00273	26280	2291	65.48997	7810	810	10.4	0.00936	3.43
21	500	-400	0.00355	26280	2291	85.16095	7810	909	11.6	0.01234	3.48
22	500	-400	0.01276	26280	2291	306.09964	7810	996	12.8	0.04492	3.52
23	200	350	0.03276	26280	2291	785.87964	7810	1024	13.1	0.11581	3.54
24	100	200	0.07971	26280	2291	1912.16319	7810	1008	12.9	0.28112	3.53

SAN BERNARDINO - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.82487	26280	2291	43776.80643	7810	945	12.1	6.37681	3.49
2	50	50	2.67182	26280	2291	64094.28998	7810	857	11.0	9.21822	3.45
3	50	50	3.50148	26280	2291	83997.00372	7810	768	9.8	11.92800	3.41
4	50	50	4.30472	26280	2291	103265.92808	7810	659	8.4	14.44077	3.35
5	50	100	5.06866	26280	2291	121592.08474	7810	547	7.0	16.74130	3.30
6	100	100	5.91978	26280	2291	142009.60242	7810	433	5.5	19.25032	3.25
7	100	100	6.91876	26280	2291	165974.13364	7810	332	4.3	22.19499	3.21
8	100	100	7.75458	26280	2291	186024.61962	7810	229	2.9	24.53827	3.16
9	100	100	8.257	26280	2291	198077.17300	7810	160	2.0	25.89244	3.14
10	100	100	8.0456	26280	2291	193005.89840	7810	125	1.6	25.11463	3.12
11	100	100	7.431	26280	2291	178262.25900	7810	116	1.5	23.16900	3.12
12	100	100	6.66787	26280	2291	159955.53343	7810	132	1.7	20.83297	3.12
13	100	100	5.82847	26280	2291	139819.16683	7810	171	2.2	18.30333	3.14
14	100	100	4.91446	26280	2291	117892.98094	7810	227	2.9	15.54701	3.16
15	100	100	3.97902	26280	2291	95452.71078	7810	302	3.9	12.71347	3.20
16	100	100	2.9845	26280	2291	71595.17050	7810	393	5.0	9.65285	3.23
17	100	100	1.96987	26280	2291	47255.21143	7810	483	6.2	6.44946	3.27
18	150	150	1.16932	26280	2291	28050.81748	7810	591	7.6	3.88569	3.32
19	250	300	0.63256	26280	2291	15174.48184	7810	703	9.0	2.13515	3.38
20	400	500	0.28079	26280	2291	6735.87131	7810	810	10.4	0.96227	3.43
21	400	500	0.14007	26280	2291	3360.13923	7810	909	11.6	0.48691	3.48
22	150	-100	0.19283	26280	2291	4625.79887	7810	996	12.8	0.67887	3.52
23	50	100	0.50387	26280	2291	12087.33743	7810	1024	13.1	1.78122	3.54
24	50	50	1.0492	26280	2291	25169.25880	7810	1008	12.9	3.70027	3.53

SAN BERNARDINO - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	60.43292	26280	2291	1449725.31788	7810	945	12.1	211.17630	3.49
2	0	50	69.41259	26280	2291	1665138.62151	7810	857	11.0	239.48492	3.45
3	0	50	77.69048	26280	2291	1863716.92472	7810	768	9.8	264.65733	3.41
4	0	50	85.534	26280	2291	2051875.12600	7810	659	8.4	286.93541	3.35
5	0	50	93.35436	26280	2291	2239477.74204	7810	547	7.0	308.34060	3.30
6	0	50	100.18756	26280	2291	2403399.37684	7810	433	5.5	325.79631	3.25
7	50	50	106.42361	26280	2291	2552995.98029	7810	332	4.3	341.40091	3.21
8	50	50	125.22838	26280	2291	3004103.60782	7810	229	2.9	396.26746	3.16
9	50	50	150.67387	26280	2291	3614515.46743	7810	160	2.0	472.48568	3.14
10	50	50	184.43774	26280	2291	4424476.94486	7810	125	1.6	575.72895	3.12
11	50	50	211.62126	26280	2291	5076582.40614	7810	116	1.5	659.81055	3.12
12	50	50	232.56731	26280	2291	5579057.19959	7810	132	1.7	726.62897	3.12
13	50	50	246.19103	26280	2291	5905876.61867	7810	171	2.2	773.12169	3.14
14	50	50	248.55743	26280	2291	5962644.18827	7810	227	2.9	786.31731	3.16
15	50	50	246.83969	26280	2291	5921437.32341	7810	302	3.9	788.68371	3.20
16	50	50	238.7665	26280	2291	5727769.56850	7810	393	5.0	772.24883	3.23
17	50	50	227.65219	26280	2291	5461148.38591	7810	483	6.2	745.34576	3.27
18	50	50	209.04015	26280	2291	5014664.15835	7810	591	7.6	694.64803	3.32
19	50	50	182.12183	26280	2291	4368920.57987	7810	703	9.0	614.73485	3.38
20	50	100	150.39433	26280	2291	3607809.58237	7810	810	10.4	515.40137	3.43
21	50	100	130.14718	26280	2291	3122100.70102	7810	909	11.6	452.41280	3.48
22	50	100	105.33813	26280	2291	2526956.40057	7810	996	12.8	370.84773	3.52
23	50	100	85.36188	26280	2291	2047746.13932	7810	1024	13.1	301.76041	3.54
24	50	100	68.96638	26280	2291	1654434.48982	7810	1008	12.9	243.22765	3.53